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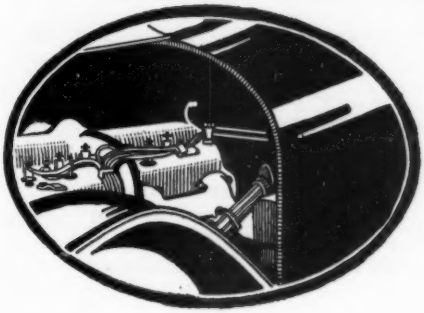

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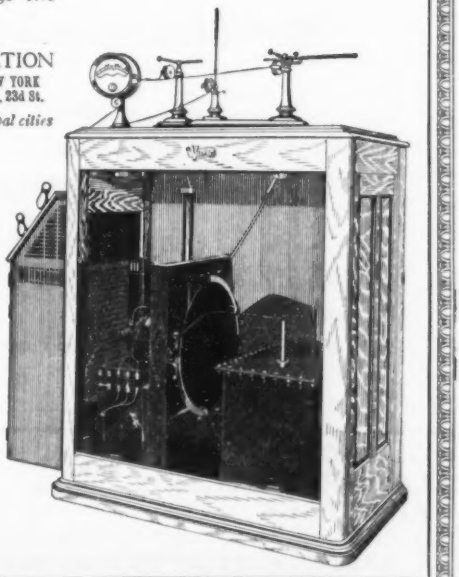

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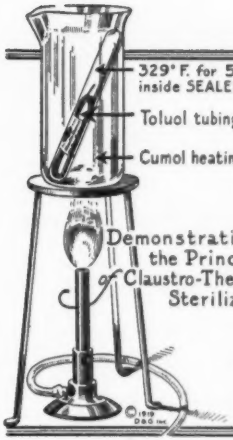
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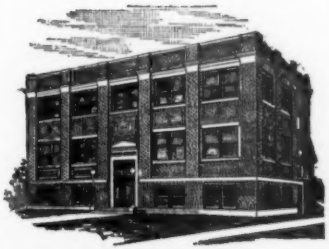

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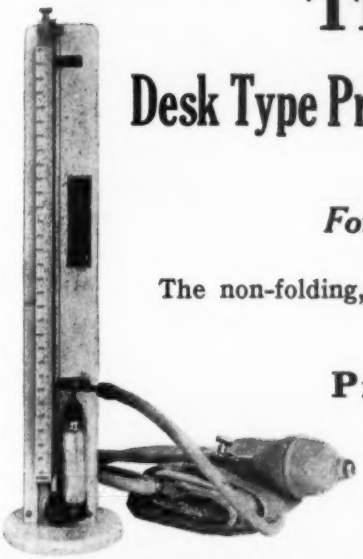

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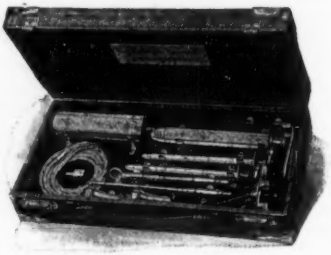

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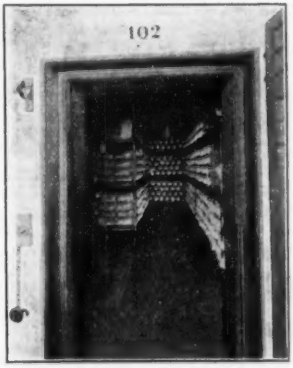

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MARCH, 1920

ORIGINAL ARTICLES

THE PHYSICAL SIGNS OF FOREIGN BODIES IN THE BRONCHI.[1]

By Thomas McCrae, M.D.,

PROFESSOR OF MEDICINE IN THE JEFFERSON MEDICAL COLLEGE, PHILADELPHIA.

Through the kindness of my colleague, Professor Chevalier Jackson, an opportunity has been given to study the physical signs in a considerable number of patients with foreign bodies in the bronchi. This study is a discussion of the clinical aspect of some of the problems presented. No attempt is made to discuss the other phases. Dr. Jackson has done this in various publications, and a summary of many of the points can be found in a recent article.[2]

The study of these cases suggests a number of clinical problems: (1) How frequent is the occurrence of foreign bodies in the bronchi? (2) What symptoms and signs are there which are diagnostic or suggestive of their presence? (3) Is there any clinical picture or are there physical signs suggestive of the presence of a foreign body in the absence of roentgen-ray findings? (4) Are there definite physical signs suggestive of a particular kind of foreign body? (5) To what extent can we reduce the chance of error in failing to recognize the presence of a foreign body in a bronchus?

It is evident that we have to discuss very different sets of conditions, and that no one description can cover all the cases. Thus

[1] Read at the meeting of the Association of American Physicians, June, 1919.

[2] Jackson, Chevalier: Observations on the Pathology of Foreign Bodies in the Air and Food Passages, Surg., Gynec. and Obst., 1919, p. 201.

we have certain signs due to the foreign body and the local reaction set up by it, and others due to changes in the lung supplied by that bronchus. The character of the foreign body is important; a safety-pin may do little damage while a peanut causes marked changes very rapidly. One foreign body plugs a bronchus completely, another acts as a ball valve. Pus forms below the foreign body and may be retained or escape. The secretions may be forced into bronchi other than the one involved, or from the affected lung into the bronchi of the opposite one, so that signs are found on both sides. The signs may change greatly from hour to hour in acute cases so that any set description fitting all cases is out of the question.

1. THE FREQUENCY OF FOREIGN BODIES IN THE BRONCHI. It is quite impossible to give any statement on this point. In the Jefferson Hospital material they are comparatively common, but this is exceptional, as the patients come from all parts of the United States and Canada to consult Dr. Jackson. Two things suggest that such cases are more common than we have generally supposed. One is the number of patients who come with a history which shows that for months or years a foreign body has been present and unsuspected and the other is the number of patients from the near neighborhood with a foreign body, but without suspicion of it on their part. The roentgen rays have aided greatly in the recognition of certain foreign bodies, but there are substances which do not show in a roentgen-ray plate, and it is for the recognition of these that a study of the physical signs is particularly important. Certainly, the impression has been strongly forced upon me that the condition is much more common than the majority of us have suspected and my memory brings up cases seen in the past in which the presence of a foreign body was probably overlooked.

One point deserves emphasis in this connection—the history. Most of us would suppose that there would be small chance of a foreign body passing into the trachea and then to a bronchus without marked acute symptoms which could not fail to be noticed. We must realize that this is not necessarily the case and that fairly large bodies may reach the bronchi without any acute symptoms. For example, in one case (No. 611[3]) there was no definite history as to when the foreign body, which was an atomizer tip, had been aspirated. The patient knew nothing of it, but on searching his memory remembered that about eighteen months before a tip had disappeared. This particular patient had been thought to have tuberculosis. At the time of writing this paper a boy of eight years of age is under observation. He was sent to the clinic for some laryngeal condition and examination shows part of a collar-button in a bronchus. No history can be obtained of any acute

[3] The numbers are those in Dr. Jackson's series, and are given to identify the cases when reported elsewhere.

onset and there is no clue as to the time of aspiration. Such cases emphasize the need of caution in concluding that the introduction of a foreign body must cause some symptoms or disturbance which can be noted. This point cannot be too strongly emphasized.

My impression is that these unrecognized cases of foreign bodies in the bronchi are by no means rare. Naturally the greatest difficulty arises in the instances in which the body cannot be recognized by the roentgen rays, probably in from 10 to 15 per cent. of all cases. There are two sharply defined groups of unrecognized cases. The first is that in which the body sets up a very acute inflammatory process and death follows in a short time—the diagnosis of pneumonia usually being made. This is particularly likely to follow the aspiration of some variety of nut, especially a peanut, by a young child, and how many of these cases occur we have no means of knowing. The younger the patient the more acute this process usually is and in such cases it is evident that the history is often not complete. The other group is that in which marked changes are set up and the symptoms become chronic, a diagnosis of pulmonary tuberculosis or bronchiectasis being made. In such a case in an adult seen some years ago there were marked signs over the lower right lobe. We suspected the possibility of a foreign body but there was no history to suggest it and repeated roentgen-ray examinations did not aid. After eighteen months of illness she coughed up a small piece of bone. This was followed by rapid recovery.

2. Clinical Features Caused by Foreign Bodies. The greatest possible diversity occurs as the signs vary markedly. A safety-pin, particularly if closed, is not likely to cause much change in a short time while a screw or tack which plugs a bronchus results in rapid changes. One substance may plug a bronchus or set up inflammation which does the same thing, and another may entirely plug a bronchus at one time and a little later allow air to enter past it, so that it is evidently impossible to give any exact account of the signs. The foreign body is more often on the right side and has a tendency to go to the bronchi supplying the lower lobes.

The change in the signs in a short interval is a striking point. In some cases this is apparently due to a change in position of the foreign body, in others air may enter at one time and not at another. Thus in one patient the signs suggested collapse of the involved lobe and a few hours later it was apparently overdistended, suggesting a valve action of the foreign body. The amount of secretion and the effect of coughing on its expulsion may cause rapid changes in the signs. The signs may be local or general; it is not uncommon to find signs over both lungs. Emphasis should be placed on this point, as in some cases the signs are more diffuse on the unaffected side. This may lead to serious error and appears most likely to occur in the younger patients.

There is only one sign which has been present in every patient seen by me, and that is *decreased expansion* of the affected side. It may be the only sign present, as in the following case:

No. 697. The patient was a woman, aged thirty-two years, who, two weeks before, coughed while holding a safety-pin in the mouth and aspirated it. She felt some soreness in the throat, followed by a tickling sensation and a hacking cough. Two days later she had more severe coughing, followed by pain in the upper right chest. On admission she complained of slight cough and pain in the *right* side of the chest. On examination there was *only one sign* to be found—diminished expansion of the *left* side. The percussion note and the breath sounds were clear. No rales were heard. In view of this the opinion was given that the foreign body was on the left side, but that there was no clue to its position beyond that. The roentgen ray showed an open safety-pin in the left bronchus without any evidence of a pathological process in the lung beyond the pin. Dr. Jackson removed the pin and three hours afterward the expansion was absolutely equal on the two sides.

In some cases it has been possible to hear rales, which may be described as rather characteristic, over a small area. In each case in which they have been observed the foreign body was metallic, but the cases are too few to draw any positive conclusions. These particular rales may be described as very fine and softer than the early crackling rales heard in lobar pneumonia. They may be compared to the crackle made by very fine tissue paper. In the two cases given there was no other evidence of any change in the lung tissue made out by physical or roentgen-ray examination.

No. 683. The patient was a girl, aged eighteen years, who was having some dental work done on December 23, 1918. A dental root-canal reamer was placed in the tooth and the dentist turned to get another instrument. The patient felt the reamer slip out of the tooth and lodge on the tongue. She tried to take hold of it, but it disappeared. She had a paroxysmal attack of coughing but did not feel any sensation of a foreign body in the throat or air passages. Two days later she took ill with what was regarded as an attack of influenza, with which she had a severe cough and considerable sputum. After this she was very weak and had some cough, which was slightly productive. She complained of an occasional sharp shooting pain in the lower left thorax, which seemed to be brought on by coughing. On admission on January 31, 1919, examination showed slightly less expansion of the lower left thorax. The percussion note seemed equal on the two sides. The breath sounds were very feeble on both sides, and this seemed to be rather more marked over the lower left lobe. In the lower left axilla there were very fine crackling rales, which

seemed to come from a distance. They were heard mostly at the end of inspiration and were slightly more marked on deep breathing, but were never loud. There were no definite signs of any solidification in the lung tissue. The roentgen-ray study showed the foreign body in the left lower lobe bronchus. There was no evidence of change in the lung beyond it. The reamer was removed on February 1. Dr. Jackson found the trachea very red and inflamed. The left bronchus showed a high degree of inflammation, with adherent masses of thick secretion which surrounded the foreign body, part of which was in the posterior branch of the left inferior lobe bronchus.

It is certainly surprising that there were not more signs with this degree of change in the bronchus. Two days later there was still slightly less expansion of the lower left side and a very few of the fine, crackling rales could be heard on deep breathing.

No. 690. The patient, a man, aged fifty-six years, was seen on March 15, 1919. On March 7, while standing on a ladder holding a staple in his mouth, he endeavored to answer a question and aspirated the staple. He did not have any symptoms at the time. The next day he complained of tickling in the throat, and a roentgen-ray examination was made which showed the staple in a bronchus. An attempt at removal was made (in another place), but was not successful. At the time of examination the patient stated that the roentgen ray showed the staple in the *left* lung and pointed to the third left interspace as the spot where he felt it. The importance of not accepting a patient's statement as accurate was shown by the examination. This was complicated by the fact that he evidently had old trouble on the right side, and he stated that he was thought to have had tuberculosis in youth. The right shoulder was lower, there was less expansion, the right scapula was longer and moved less, and there was dulness, with harsh breath sounds at the right apex. The percussion note was equal in the two axillæ, but in the lower right axilla there were fine rales at the end of inspiration heard over a small area. These seemed to come from a distance and were extremely fine. They were the same as those heard in case No. 683. No rales were heard on the left side. Despite the positive statement by the patient that previous roentgen-ray studies had shown the staple in the left lung and that he could feel it there, these rales suggested that it was in the right lower lobe bronchus. The roentgen-ray study (Dr. Manges) showed a metal staple in the main bronchus of the lower right lobe and very little pathology in the lung below the staple. Dr. Jackson found the staple in this situation and removed it. The fine crackles were heard unchanged three hours after the removal, but had disappeared the following day.

One hesitates to give too much importance to these rales, but they are different from rales which I have heard in any other condition,

FIG. 1.—Case 690. Staple in right lower lobe of bronchus. Little change below the foreign body.

and have only been heard in cases in which the foreign body was metallic in character. They are certainly worth keeping in mind,

but it is well to state that very careful listening is required to hear them. They might easily be missed in a hurried or perfunctory examination.

The next stage of the development of the signs is found when changes have occurred in the lung. An example is as follows:

> No. 684. This patient was a boy, aged three years, seen on January 31, 1919. The history was that on December 21, 1918, it was noticed that the patient had something in his mouth which was suddenly aspirated. A severe coughing attack followed and the patient was blue for a time. In the next two or three days repeated coughing attacks occurred and there was a slight elevation of temperature. The patient was attended by a physician who made a diagnosis of influenza and later of pneumonia on the left side. During this time the patient seemed to be very ill and had fever, which occasionally reached 104°, with very severe cough and considerable pain in the left side of the chest. Examination was difficult, as the child was crying constantly and was very restless. The expansion was diminished on the right side. The percussion note was clear and hyperresonant on the left side, but on the right side there was some impairment on percussion throughout, slight in the upper part and most marked in the lower right front, axilla and back. The breath sounds were clear throughout the left side and not a single rale was heard. On the right side, on quiet breathing, expiration was not heard, but was distinctly audible when forcible expiration was made. In general the breath sounds were distant over the right side. In the upper part of the right chest the breath sounds were rather higher pitched than normal, but were not tubular. There were many coarse, crackling rales in the lower right interscapular region and a certain number in the lower axilla. After forcible coughing the rales changed somewhat, becoming rather fewer. There was no wheeze heard. The signs suggested that the foreign body was in the lower right bronchus. Dr. Jackson removed a machine screw from the right inferior lobe bronchus. Large amounts of pus escaped during the operation and were coughed up subsequently.

It is interesting to note how frequently the diagnosis of pneumonia is made in these cases. This comes out very frequently in the histories, but a careful study of the physical signs should prevent this error. Fever is very common after the aspiration of a foreign body, and if there is dyspnea, with cough and bloody or blood-streaked sputum, the diagnosis of pneumonia is suggested before the examination of the thorax is made. Decreased expansion on one side and dulness may suggest the diagnosis, but a careful study of vocal fremitus and the auscultatory signs should show that if the condition is pneumonia there is plugging of the bronchus—a rare

condition. The writer has not seen pneumonia associated with any case of foreign body in a bronchus. The local signs are usually those

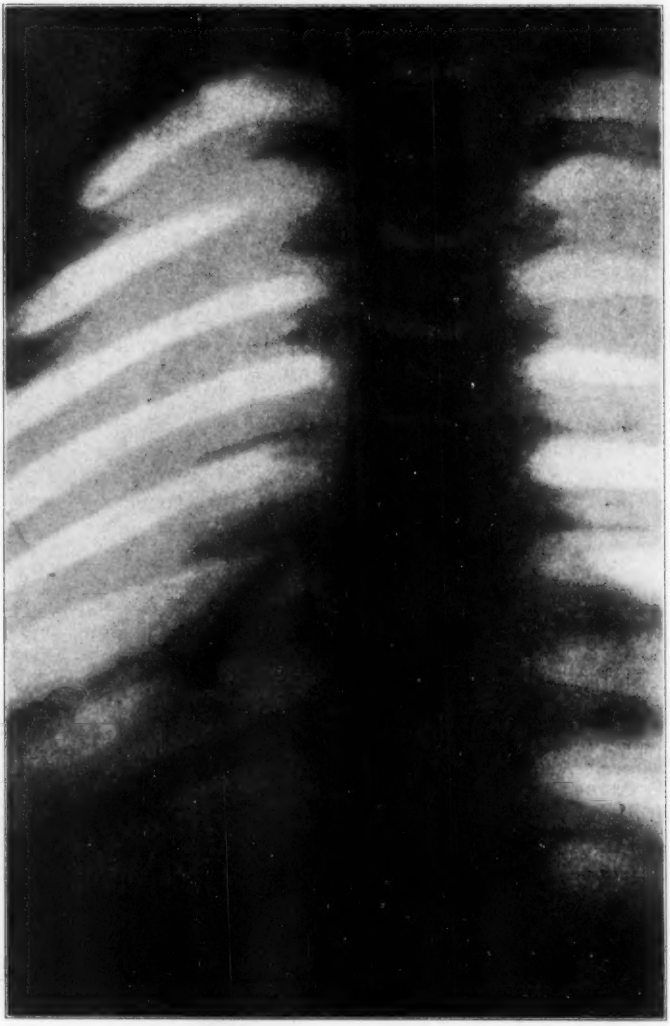

FIG. 2.—Case 684. Foreign bodies in bronchi.

due to the supplying bronchus being obstructed. Variations depend on the quantity of air in the portion of lung, the amount of

secretion and the persistence and completeness of the plugging of the bronchus. In many cases there are no breath sounds or rales heard over the affected parts, in others there may be rales but no breath sounds. In the majority the affected portion of lung seems to be airless and in the roentgen-ray plates at early stages shows as a homogeneous shadow. Later fibrosis, abscess formation and bronchiectasis may be found. These show no special peculiarity in the physical signs, but are important in that they may be wrongly diagnosed as tuberculosis.

"ASTHMATOID WHEEZE." This term has been employed by Chevalier Jackson to describe a sign which is present in a considerable number of cases of foreign body in the trachea or bronchi. He has discussed it in considerable detail in a recent article.[4] This sound can be heard by placing the ear or the bell of the stethoscope close to the patient's mouth. It may be heard with both inspiration or expiration, but sometimes is only audible at the end of forced expiration. Forcible respiratory effort usually increases the loudness and intensity. It differs from the wheezing sounds heard with bronchial asthma, but this difference is difficult to describe in words. The "wheeze" varies in pitch and loudness, depending on the character of the foreign body causing it. In no case in which the writer observed it could the sound be heard over the chest wall. In some cases the sound comes and goes without any explanation being evident. The report of a case in which it was very marked is as follows:

> No. 675. The patient, a white male, aged forty-four years, was admitted on December 20, 1918, complaining of cough and shortness of breath. Four days before admission a dentist was fitting a gold bridge, when it slipped back in the mouth and was aspirated. Following this there was a paroxysm of coughing and the patient had a momentary sensation of a foreign body in the throat. The same evening he had an attack of dyspnea, which lasted for about half an hour and which he compared to asthma. With this there was an unproductive cough. With the coughing he had noted a certain amount of wheezing. On examination on December 21, 1918, the patient was propped up in bed. He had slight dyspnea but no cyanosis. On listening with the ear near the patient's mouth a wheezing sound was heard, most marked with inspiration and very slightly heard with expiration. It seemed to be rather more marked when he took a deep breath. Expansion was much less over the lower right chest. On percussion there seemed to be slightly less resonance on the right side which was most marked in the lower right axilla and back. The breath sounds in general, were rather harsh and this was more marked on the

[4] AM. JOUR. MED. SC., 1918, clvi, 625.

right side. No rales were heard on the left side, but there were a great many over the whole of the right lung. They were of very variable character, the majority whistling and groaning, and some with rather a wheezing character. The roentgen rays showed an artificial dental plate, apparently composed of three teeth in the right upper bronchus. Removal was done on December 21, and immediately after it the wheezing sounds heard at the mouth disappeared. Two days after removal expansion of the two sides of the chest seemed absolutely equal, percussion was clear, no rales were to be heard and the breath sounds seemed to be equal on the two sides.

This sign appears to be of considerable value, particularly in the cases in which the foreign body does not show in the roentgen-ray plate. It has been observed in many cases and occurs with different substances. It should be listened for in all cases of obscure thoracic diagnosis in which the presence of a foreign body has to be considered.

Certain foreign bodies set up very acute and dangerous changes, among which the *peanut* takes first place. It sets up a rapid and severe reaction and the younger the child the more severe this is. The following is an example:

No. 682. The patient was a boy, aged five years, who on December 20, 1918, choked while eating peanuts. There was much coughing and gagging, with cyanosis at the time. Later the symptoms subsided, but in the evening the father noted that there was a wheezing sound at intervals, with a dry cough. Until January 7, 1919, the patient seemed well and played as usual. On this date the cough increased in severity and there was severe dyspnea, but the wheezing ceased. The physician said that air was not entering the left lung. On January 14 an attempt was made to do bronchoscopy (in another city), but without success. The patient was admitted to the Jefferson Hospital on January 16, 1919. On examination he was comfortable and showed neither distress nor cyanosis. Over the right side there were harsh breath sounds, with many medium and coarse rales. The left side of the thorax showed decided fulness, but no expansion. Vocal fremitus was difficult to obtain, but seemed much decreased. The percussion note had a curious quality of tympany, with an element of dulness. The breath and voice sounds were not heard over the left side. No rales were heard. The heart was not displaced. We considered that the peanut had acted as a ball valve and allowed air to enter but not to escape. The roentgen ray confirmed this, and after the removal of the peanut showed an equal amount of air in the two lungs.

The larynx was found to be much swollen and the trachea greatly inflamed. There was pus coming freely from the right

main bronchus. The left bronchus was intensely red and swollen and contained tenacious pinkish pus. After sponging this away the peanut was seen in the left bronchus. It was removed without crushing. After removal the left side still showed decreased expansion, but harsh breath sounds could now be heard, with many loud bubbling rales. Within four days the child felt well and the signs in the chest had nearly cleared. There was still less expansion, with the percussion note and breath sounds about normal.

This case illustrates one point which may cause error—the presence of signs on both sides. This is usually due to secretions from one side being forced or carried over to the opposite bronchus. Occasionally a foreign body may be moved from one side to the other, or, in the case of a nut portions may be broken off and carried over. It may be said, however, that there is rarely any doubt of the side involved if a careful examination is made. The decreased expansion is the most important guide in deciding this point. Special reference must be made to the need of care in the peanut cases, as a general process is set up in both lungs and the roentgen-ray plate may show more pathological changes on the side which does not contain the nut.

To this condition the term *Arachidic Bronchitis* has been applied by Jackson and Spencer.[5] They apply this term to the condition set up by the presence of a peanut in the respiratory tract, but other nuts give much the same symptoms. The clinical picture is rather a distinctive one associated with an edematous, purulent tracheobronchitis which often results in lung abscess. The cases appear only in children and the symptoms come on rapidly, with high fever, severe toxemia and marked thoracic signs. The dyspnea is extreme, cyanosis is usually marked and there may be a purulent tenacious sputum. The fever is usually irregular, but is high for part of the day. The signs are general and are those of an intense general bronchitis, with a great variety of rales, the majority coarse and bubbling. There is decreased expansion on one side as a rule, but if portions of the nut are on both sides, as sometimes happens, this does not hold. Depending on the size and situation of the particles there may be dulness and absence of breath and voice sounds if a bronchus is plugged. The lung supplied by the plugged bronchus contains much secretion and has been described as "drowned lung." This is often mistakenly regarded as representing pneumonic consolidation. The "asthmatoid wheeze" is present in a majority of the cases. The voice is usually lost.

"We should always remember the possibility of peanut bronchitis when consulted regarding a child who rather suddenly develops irregular fever, restlessness, dyspnea with cyanosis, paroxysmal

[5] Jour. Am. Med. Assn., 1919, lxxiii, 672.

cough and the signs of a diffuse, generalized bronchitis, attended with wheezing respiration. A history of choking on a peanut, or of eating peanuts at about the time of inception of the illness, renders the diagnosis almost certain." (Jackson and Spencer.)

3. As regards the question of distinctive signs of foreign bodies one speaks with great hesitation. The two signs mentioned certainly deserve careful study, namely, the presence of the fine "tissue-paper" rales over a small area and the "asthmatoid wheeze.' The former has been observed with small metallic bodies only, but further observations may show that it occurs with others. They are not necessarily present in every case of small metallic bodies. The "wheeze" seems to offer more as a suggestive sign; Dr. Jackson has now observed it in over 50 cases, so that it seems to be of much importance. I venture to suggest the value of keeping it in mind and noting its presence or absence in doubtful cases.

In the very acute cases, usually due to some substance such as a nut or burr, the marked general signs may be of value, especially if there is any history. The problem is complicated by the presence of fever, which may be high, and the general picture of an acute infection. Some of these patients present a picture of the most intense toxemia. A general picture suggestive of a severe bronchopneumonia, but without any positive physical signs of it, should excite suspicion of something else.

The occurrence of signs in a lower lobe for which there is no evident explanation should always suggest the possibility of a foreign body. The same may be said to be true of local signs anywhere the etiology of which is not clearly proved.

4. As regards physical signs suggestive of a particular kind of foreign body, in the absence of a positive history one hesitates to speak with any certainty. One might *suspect* but it seems difficult to be *sure*. Evidently this is most important in the cases in which the roentgen rays give no aid. In such instances the acute general signs caused, for example, by a peanut are perhaps as suggestive as those of any group.

5. The Reduction of Errors in Diagnosis. It is evident that errors will be fewer if the possibility of a foreign body is considered. Too often it is not thought of and a hasty examination, soon after the entrance of a foreign body, results in a careless diagnosis, usually of pneumonia, which is not revised. This seems the only explanation when the changes due to a foreign body are diagnosed as tuberculosis, and this is accepted for years. In general, foreign bodies tend to go downward and set up changes in the lower lobes. It seems to take a great deal of telling to convince some of us that tuberculosis at the base of a lung alone is very rare. In every case of basal involvement, in every case of abscess and of bronchiectasis the possibility of a foreign body should be considered.

In the acute cases, typically illustrated by the reaction to a

peanut, the problem may be more difficult, but the suggestions given by Jackson and Spencer are an aid. The picture is in some ways like a very acute severe bronchitis with bronchopneumonia, but it reads more like it in the description than it looks like it in the patient. It is one of the differences which is difficult to state in words, but is suggested at once after one has seen the patients. The intense toxemia, marked dyspnea and cyanosis, and high fever without any signs of pneumonia, are points to be noted.

SUMMARY. 1. Cases of foreign body in a bronchus are not mere curiosities but are more common than we have supposed.

2. There may be no disturbance at the time of entrance of a foreign body and no suggestion in the history of such a happening.

3. Certain signs are of value, especially decreased expansion on the affected side, the presence of very fine rales and the "asthmatoid wheeze."

4. Some foreign bodies, such as a peanut, set up a very acute general process which is fairly distinctive. Other structures, such as metallic objects, cause permanent changes, usually in a lower lobe.

5. The chief errors in diagnosis are to mistake the signs for those of pneumonia in the early stages and in the acute cases, and for tuberculosis after the body has been present for some time.

THE OCULOPUPILLARY FIBERS OF THE SYMPATHETIC SYSTEM: DIVISION OF THE FIRST THORACIC ROOT IN MAN.[1]

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IT is surprising that so little is known regarding the course of the sympathetic fibers in the brain; especially is this true of the cerebrum. We have evidence as to the location of the oculopupillary fibers of the sympathetic system in the cervical cord, medulla oblongata and pons, but scarcely anything is known of these fibers in their relation to higher parts of the brain. My attention has been directed at this time only to the oculopupillary fibers of the sympathetic system. Paralysis of these fibers produces the Claude Bernard-Horner syndrome. It is not definitely known that this paralysis occurs with hemiplegia of cerebral origin, but a case in which it seems to have so occurred has come under my observation and has led to investigation of the literature bearing on this subject.

[1] Read before the Philadelphia Neurological Society, November 21, 1919.

W. C., aged eleven years, was examined October 30, 1919.

When six months old he had a series of convulsions. The attack lasted about two weeks. When he began to walk at two years of age his mother noticed that he was awkward and could not use his left arm and hand and that the left leg was weak. Between the ages of six months and two years his mother said he was "helpless," *i. e.*, he was not as active as other children. At the time of examination he had been in school five years, was backward and was only in the second grade.

He had left hemiplegia, implicating also the face. The tendon-jerks were exaggerated on the left side and the Babinski reflex was present on the left side. The left pupil was smaller than the right and the mother was unable to say how long this difference had existed, but evidently it had been present a long time. The left palpebral fissure was smaller than the right. Ocular movements were normal. The left upper and lower limbs were not as well developed as the right. The tongue when protruded deviated to the left. The left foot was in the position of talipes equinus.

Dr. H. M. Langdon reported on the examination of the eyes. Vision: O. D., 6/12. O. S., 6/12?? Right palpebral fissure 10 mm., left 7½ mm. Right pupil 3 mm., left 2½ mm. Exophthalmos, right eye, 14 mm., left 13½ mm. Ophthalmoscope showed disks oval, good color, with no fundus changes.

There was evidently a paralysis of the cervical sympathetic on the side of a long-standing hemiplegia, as shown by the small pupil, small palpebral fissure and retracted eyeball. No evidence of injury of the neck and no enlargement of the thyroid gland causing pressure on the cervical sympathetic could be found. There was no cranial nerve palsy suggesting a basal lesion. The lesion must have been above the lower part of the cerebral peduncles or some cranial nerve probably would have been involved. There is no evidence that the sympathetic palsy occurred simultaneously with the hemiplegia, but there was no other cause known than that producing the hemiplegia, and the mother could not state when the oculopupillary symptoms developed; they had been present a long time according to her statement.

In the discussion of this case Dr. S. D. Ingham, described a case in which he had made the diagnosis of a thalamic lesion, because of disturbance of tactile pain and thermic sensations, with spontaneous pain of unilateral distribution. Associated sympathetic symptoms consisted of a small pupil and narrow palpebral fissure on the affected side and unilateral sweating of the face and trunk.

Dr. Samuel Leopold also mentioned a case he had presented in 1916, in which right hemiplegia with Jacksonian epilepsy confined to the upper extremity was associated with oculopupillary symptoms.

Examination of the literature has revealed some interesting information. Karplus and Kreidl, by placing an electrode at the

base of the brain in the living cat, obtained important findings. The point of irritation was just behind the optic tract near the oculomotor nerve lateral to the infundibulum. Maximal dilatation of both pupils resulted from this irritation, with widening of both palpebral fissures. This result was constant in more than twenty cats, whether the animal was in a state of narcosis or not. A weak current was sufficient to cause the dilatation. When a very feeble current was used the effect on the heterolateral eye was always prompter than on the homolateral eye.

They demonstrated that this irritation was conveyed through the cervical spinal cord. The cervical sympathetic on each side was exposed. Irritation in the above-mentioned area of the base of the brain was then made to cause dilatation of the pupils. If after this irritation the heterolateral cervical sympathetic was cut, renewed irritation of the base of the brain caused no effect on the heterolateral side, while the effect on the homolateral eye was as prompt as it had been previously. If then the homolateral cervical sympathetic was divided the pupil of the homolateral side could not be influenced by basal irritation. These experiments were repeated, with the same results in a number of animals.

Electrical irritation of the dura, the cerebral hemispheres, the peduncles and the infundibulum was without effect.

When the intracranial portions of the trigeminal and oculomotor nerves and the optic tract were cut, irritation of this mentioned area of the base of the brain produced the same results as before. Removal of the cerebral hemisphere, even complete separation of all parts of the cerebrum from this irritated area, so that only a little of the cerebrum remained with the brainstem, did not disturb the effect of the irritation.

They concluded that irritation of the base is transmitted through the homolateral cerebral peduncle and crosses in part lower down to descend in the cervical cord, predominatingly in the heterolateral side and then through both cervical sympathetic cords to the eyes.

They did not know whether a center was irritated in these experiments, and if so possibly it was the corpus subthalamicum; or whether a tract near the surface was irritated, but they inclined to the view that a center was irritated. They proposed to study, further, the relation of their basal irritable zone to the cortex.

It seems probable from these experiments that sympathetic fibers for the eye exist in the cerebral peduncle in the cat and that most of these fibers supply the heterolateral eye.

In their second paper on this subject, Karplus and Kreidl stated that their experiments were made on dogs as well as cats. They believed the results obtained by them, even after removal of the cerebral mantle, prove that the irritable basal area is a center and not a place where sympathetic tracts come near the surface. They extirpated the cerebral hemisphere on the side in which they

irritated the basal part of the brain and they did not think it necessary to extirpate the contralateral cerebral hemisphere. They believed there was no indication of decussation between the cerebral cortex and the midbrain. In this second paper they demonstrated that a certain relation exists between the cerebral peduncle and the homolateral cerebral hemisphere and not the heterolateral hemisphere.

They determined by marking the irritable zone at the base and then making microscopic sections that their sympathetic center was in the hypothalamus near the dorsomedial portion of the foot of the cerebral peduncle, in the frontal part of the corpus subthalamicum.

They obtained similar results in the ape, but the effect on the contralateral sympathetic was even greater than in the cat.

They found that in cats they were able frequently to obtain by electrical irritation of one frontal pole of a cerebral hemisphere, with a weak current, bilateral maximal irritation of the cervical sympathetic. They then destroyed their basal sympathetic center on the side on which they had irritated the frontal lobe and were no longer able to obtain irritation of the cervical sympathetic cords by placing the electrode on the frontal lobe in the region from which they had previously been successful. Irritation of the corresponding spot in the other frontal lobe still produced irritation symptoms in both cervical sympathetics. This is further evidence that the basal area is a center.

In their second paper Karplus and Kreidl referred to the fact that v. Bechterew obtained the signs of irritation of the sympathetic to the eye by stimulation of the medial portion of the optic thalamus, but they stated he did not determine whether he stimulated a sympathetic center or a tract in the part stimulated. This experiment may have some bearing on the case reported by Ingham referred to above.

Karplus and Kreidl also reported in their second paper that stimulation of the sympathetic to the eye by pain depended on the hypothalamic center they described.

In their third paper from their experiments on cats they concluded that each half of the cervical cord conducts impulses from the brainstem to both cervical sympathetic cords. Decussation does not occur in the cervical spinal cord but below the cervical cord. They acknowledge that their results from cats are at variance with those of others from experiments on rabbits. I feel certain that in man the decussation of these fibers is not in or below the cervical spinal cord.

Different investigators have represented different parts of the cerebral cortex as suitable for electrical irritation of the cervical sympathetic, but Karplus and Kreidl used the frontal lobe. They believed it is certain that in the cat the hypothalamic region as

well as the frontal cortex sends impulses to both cervical sympathetic cords. The irritation of the hypothalamic region always affects the cervical sympathetic, but the irritation of the cerebral cortex in different parts is not always shown in the cervical sympathetic. They agree with other authors that there is no definite sympathetic center in the cerebral cortex, it is certain only that in many animals a feeble current may more readily cause irritation of the cervical sympathetic from certain parts of the cerebral cortex than from other parts. This is not a very elucidative statement and it is evident that very little is known of sympathetic fibers in the cerebrum.

Their work seems to establish the existence of an important sympathetic center in the hypothalamus, but there is still much doubt as to how the fibers from this center get to their point of exit from the spinal cord, certainly the doubt is very great as regards man. It would seem that in the cat the hypothalamic center has more control on the heterolateral eye than on the homolateral eye, and this influence is even greater in the ape than in the cat. Their work leaves us in doubt as to a decussation of oculopupillary fibers in the cerebral peduncles, but it indicates that there is no decussation of these fibers above the cerebral peduncles.

Trendelenburg and Bumke stated that extensive destruction of the cerebrum, especially of the frontal lobe, has been observed to cause a difference in the pupils, but findings are conflicting as to whether the homolateral or heterolateral pupil is contracted. They reported some experiments in which the entire cerebral mantle in cats and dogs was removed in order to determine the influence of the cerebral cortex over the sympathetic to the eye. In four experiments unilateral removal of the cerebral mantle caused slight homolateral narrowing of the pupil, but they did not determine whether this influence was exerted through the oculomotor nerve or the cervical sympathetic. The difference in size of the two pupils was slight, less than in unilateral section of the medulla oblongata or of the cervical spinal cord, and therefore they said that this control of the pupil from the cerebrum is not to be accepted unreservedly as transmitted through the cervical spinal cord on the same side. They believed the cerebral mantle has no essential influence on the control of the pupil exerted by the cervical spinal cord, as demonstrated by one of their experiments. Both cerebral hemispheres in a cat were removed, one after the other, and then unilateral section was made at the junction of the medulla oblongata and cervical cord. The homolateral pupil after the section of the cord became smaller, although before the section it had been a little larger. Their work therefore affords little evidence of the control of the cerebrum over the sympathetic fibers to the eye.

Bumke asserted that irritation of the sympathetic fibers to the iris from cortical conditions although much discussed, has not been

demonstrated, and such an occurrence is improbable. He stated that we do not know the origin of the sympathetic fibers for the eye, but that without doubt there is a relation of the cerebrum to the sympathetic innervation of the iris.

Oppenheim observed a vasomotor form of Jacksonian epilepsy, consisting of attacks of cyanosis of one arm and of the face on the same side as the affected arm, with or without unconsciousness. The cyanosis of these parts finally became persistent and was associated with myosis and narrowing of the palpebral fissure and paresis of the arm and face. The report of this observation is very brief and the context implies that the symptoms were all on the same side of the body, and the case is confirmatory of the findings in my case.

Oppenheim believed that certain of the sympathetic symptoms of the eye may be dependent on the cerebral cortex. v. Monakow stated that v. Bechterew reported a case in which the right pupil could be dilated voluntarily almost to the maximal degree. The dilatation persisted after the impulse had ceased and disappeared after winking. v. Bechterew cited another case in which both pupils could be dilated voluntarily. v. Monakow believed these observations demonstrate that a close connection exists between the cortex and the center for the dilatation of the pupil.

I have come to the conclusion that in man the oculopupillary fibers do not decussate, or at least in very slight degree, in the pons or below this in the medulla oblongata or cervical cord. In two cases of tubercle of the pons, which is a lesion destroying the axis-cylinders where it exists and not permitting them to pass through as does glioma, the oculopupillary symptoms were on the side of the lesion. I have repeatedly seen oculopupillary paralysis of the sympathetic on the side of the lesion resulting from occlusion of the posterior inferior cerebellar artery. This occlusion produces softening in the lateral part of the medulla oblongata. It is possible that the sympathetic fibers decussate in the cerebral peduncle, but the evidence of this is far from conclusive.

In the case of tubercle of the pons reported by Potts and myself the pupil was small on the side of the lesion. The left side of the pons was much larger than the right. In its upper part, just above the entrance of the fifth nerve, a tumor was found occupying the left half of the tegmentum and invading a little the pyramidal tract on the same side. In the lower part of the pons the tumor implicated the left middle cerebellar peduncle and the pyramidal fibers on the left side, but did not invade the dorsal part of the tegmentum. The right side of the pons was not encroached upon by the tumor at any part. The nucleus of the sixth and probably that of the seventh nerve escaped, but the fibers of these nerves within the pons must have been compressed by the tumor. In this case the pupils were unequal, the left being the smaller, *i. e.*, the small pupil was on the side of the lesion confined to the pons.

In another similar case studied and reported by me the same relations existed. A tubercle was found in the left side of the pons. The left cerebral peduncle was larger than the right and the tumor extended into the lower part of the left cerebral peduncle. In the middle portion of the pons it invaded slightly the right side of the pons. The left pupil was 2 mm. in size and the right 3 mm. in size. Both pupils reacted to light and in accommodation and convergence. Under homatropin the right pupil was dilated to 6 mm., the left to 4½ mm. Here also there was sympathetic paralysis on the side of the lesion in the pons and homatropin dilated the left pupil considerably less than the right. From these two cases it seems that the sympathetic fibers of the eye may be paralyzed by a lesion of the pons and that these fibers do not decussate in or below the pons.

There is scarcely any doubt as to the point of exit of the oculopupillary fibers from the spinal cord. The work of Claude Bernard and later that of Mmes. Dejerine and Klumpke have established the fact that these fibers must leave the cord by the first thoracic root, and possibly by the eighth cervical and second thoracic roots, and probably through the anterior divisions of these roots. While this determination has been made by experiments on animals and clinical observations on man, there is no case recorded, so far as I know, in which the first thoracic root alone was divided in man at operation and the resulting paralysis determined. The opportunity of determining the paralysis and the oculopupillary symptoms following division of the first thoracic root has recently been given me. At an operation for removal of a tumor of the cord it became necessary to divide this root on one side in order to excise the tumor. Oculopupillary symptoms had not existed previous to the operation, but they became very pronounced following the operation.

In the Klumpke paralysis oculopupillary symptoms form an essential part of the clinical picture. Dejerine describes the Klumpke type of paralysis as follows: It usually is preceded by a total radicular brachial plexus palsy. It assumes the form of ulnar palsy and implicates the small muscles of the hand, the thenar and the hypothenar eminences and the interossei muscles. Anesthesia of radicular type implicates the internal half of the hand and the forearm, *i. e.*, it is in the zone of cutaneous distribution of the ulnar and internal cutaneous nerves. Associated with this are myosis, narrowing of the palpebral fissure and retraction of the eyeball. She attributes the symptom-complex to a lesion of the first and second thoracic roots or of the eighth cervical and first thoracic roots.

Mmes. Dejerine and Klumpke found in sectioning the roots of the brachial plexus in the dog that only the division of the inferior roots, and especially of the first thoracic root, caused oculopupillary phenomena. Division of the eighth cervical, and especially of the

first thoracic root, made near the vertebral column, produced the same oculopupillary signs. These signs occurred in her experiments only when the first thoracic root was divided, but the eighth cervical also was divided.

In quoting Claude Bernard she states that his experiments first showed (1862) that the oculopupillary fibers pass by the first and second thoracic roots, but the predominance of the first thoracic in the symptom-complex was recognized by him. Her own experiments showed that division of the nerves of the brachial plexus causes oculopupillary phenomena only when the first thoracic is implicated. It does not seem to be possible from these experiments to exclude the eighth cervical and second thoracic roots from participation in oculopupillary function.

The lesion may not only implicate the first thoracic root in the Klumpke form of paralysis but the symptoms may indicate that the spinal cord at this level also is affected, and in such an occurrence symptoms of implication of the lower limb on the same side will be obtained. Such a case I have had the opportunity of studying.

E. C., a boy, aged nine years, was referred to me by Dr. C. H. Frazier, October 25, 1917. He had an accident February 17, 1917. While on roller skates he went off the sidewalk, chasing another boy, and ran into a wagon in the street and was thrown against the curb of the sidewalk. When he was found he had a ring about his neck of bruised tissue. The right clavicle was much fractured. The right lower limb was weak about two months and would give away in walking. After this the lower limb gradually recovered normal power. He had never had any pain in the right upper limb except at the setting of the clavicle. He had some pain at first in both lower limbs, lasting about one week. He had never had any loss of sensation in the right upper limb. The right upper lid drooped greatly and the right pupil was much smaller than the left. The right side of the face seemed smaller than the left. He lost two nails on the right hand and the nail of the right big toe after the accident.

In my examination I found the right biceps jerk was prompter than the left. The right triceps jerk was not distinct. He had normal or nearly normal power at the right shoulder and right elbow, but only a little power at the right wrist, no power in the flexor carpi ulnaris muscle and no power in the right fingers. The right arm above the elbow and the right shoulder muscles were not at all atrophied, the right forearm was smaller than the left and the right hand was intensely atrophied. The right palpebral fissure was much smaller than the left and the right pupil was distinctly smaller than the left and the right eyeball was not so prominent as the left. The power in the right lower limb was equal to that of the left. He stood well on either foot alone, but had distinctly positive Babinski, Chaddock and Oppenheim reflexes on the right

side and not on the left side. There was no change in touch, position, heat, cold or pain sensations in either upper or lower limb.

In dorsal flexion of the right hand there was weakness on the ulnar side, probably from lack of tone in the flexor carpi ulnaris muscle.

The case in which oculopupillary symptoms resulted from division of the first thoracic root, to which I have alluded, described very briefly except for these symptoms, was referred to me by Dr. P. H. Swartz, of Towanda, and is as follows:

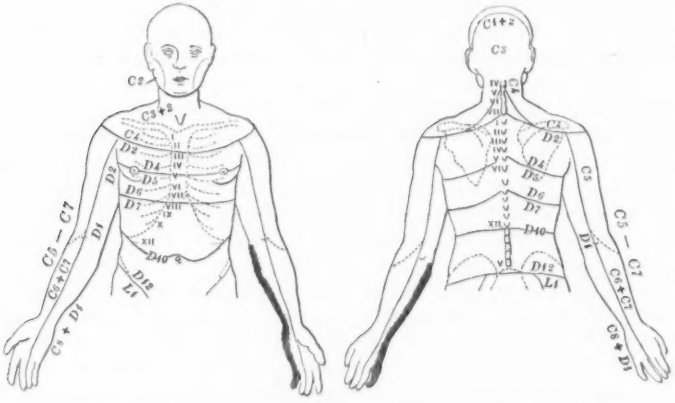
H. B., female, aged eighteen years, had complete paralysis of motion and sensation in the lower limbs and pain radiating in the lower inner side of the left upper limb to the hand, more in the little finger. The left upper limb was a little weak. The thenar and the hypothenar eminence appeared a little flattened in both hands and more distinctly so in the left hand. The fingers of the left hand were held in partial flexion at the second and third phalanges. There were no oculopupillary signs before the operation. The operation was performed by Dr. C. H. Frazier, October 18, 1919.

The spinous process of the seventh cervical vertebra was identified the day previous to the operation by the roentgen rays. With this point approximately as the center of the incision the laminæ of the first thoracic and sixth and seventh cervical vertebræ were removed. This exposure, according to the diagram in Dejerine's *Sémiologie des Affections du Système Nerveux*, second edition p. 615, should reveal the seventh and eighth cervical and first and second thoracic segments.

A tumor was found on the left side of the spinal cord in the upper thoracic region and a large posterior root passed nearly across the middle of the tumor. The tumor had much the size and shape of an almond and was cystic. There was marked difference in size between this root and the one immediately below it, as I was able to determine by careful observation, such a difference as is commonly seen between the first thoracic and second thoracic roots. It was necessary to divide this large root near its exit through the dura, as it was involved in the tumor mass, and both the anterior and posterior portions were cut. As far as could be determined by most careful observation the root cut was the first thoracic root and the symptoms resulting justified this conclusion. Following this operation the left palpebral fissure was distinctly smaller than the right and the left pupil was smaller than the right, but both reacted promptly to light and in convergence and the left eyeball was not quite so prominent. During the first few days following the operation sweating was very pronounced on the right side of the face while the left side of the face remained dry, but sweating occurred freely on both sides of the chest, so that the dryness of the left side of the face was the pathological condition. The right side of the face was more flushed than the left.

Following the operation numbness was experienced in the left little finger, the ulnar side of the ring finger, and the ulnar side of the left forearm. This at first was pronounced and was greatest in the little finger, but gradually became less intense, although it did not disappear.

The root that was cut could have been only the eighth cervical or first thoracic, and I believed it to be the first thoracic from its size and relations and the resulting numbness of the ulnar side of the left forearm and hand. The resulting palsy of the sympathetic to the eye was pronounced from the cutting of this one root, and this root evidently contained most of the sympathetic fibers to the eye. The operation was done October 18, 1919, and the palsy of the cervical sympathetic had not changed when the patient left the hospital November 11, 1919.



Showing the area of diminished sensation after division of the first thoracic root. The disturbance was greater in the hand than in the forearm.

She returned December 8, 1919. An examination made December 12 showed that touch, pain, heat and cold sensations were diminished in a narrow band on the ulnar side of the left forearm to within about two or three inches of the elbow and on the ulnar side of the hand, including the little finger. This was only a mild impairment for any form of sensation; a little greater irritation of any of these forms of sensation was promptly and correctly recognized. The impairment was greater in the hand and little finger than in the forearm. The paresthesia had disappeared from the forearm and hand in the area of slightly impaired objective sensation, where it was pronounced immediately after the operation. It persisted in all three segments of the left little finger, but sometimes was felt only in the most distal segment. There was still a

little flattening of the left hypothenar eminence and of the interosseous spaces, but this condition existed before the operation. The thenar eminence was not atrophied. I could not notice any increase of wasting, and the patient believed the left hand was becoming progressively stronger. When the left hand was at rest there was still a tendency to flexion of the fingers at the second and third phalangeal articulations, progressively increasing from the index to the little finger, *i. e.*, the hand had a tendency to assume the position of ulnar palsy. She could fully extend all the segments of the fingers of the left hand without difficulty. The grasp of the left hand was a little weaker than that of the right. Improvement in the lower limbs was very pronounced, and there was possibly a little improvement in the sympathetic supply of the left eye.

Dr. E. A. Shumway reported that when he examined the patient in a position facing the window on a rather cloudy day on December 13, 1919, almost two months after the operation, the right pupil measured 4.5 mm. and the left pupil 3.5 mm. The right palpebral fissure measured 11 mm. and the left 10 mm. in width. No difference in position of the eyeballs could be detected by the exophthalmometer.

Two instillations of a 4 per cent. cocain solution were made in the left eye, with a resulting well-marked dilatation of this pupil, so that there could not have been a complete sympathetic paralysis from division of the first thoracic root, and this would indicate that the first thoracic is not the only root conveying oculopupillary fibers, even though it may convey most of them. Cocain is believed to stimulate the sympathetic fibers to the iris.

I have found a few reported clinical cases believed to be the result of lesion of the first thoracic root without implication of oculopupillary fibers.

In the cases of uniradicular palsy implicating the first thoracic root reported by E. Farquhar Buzzard there was present a partial atrophic palsy of certain muscles of the hand and forearm, and although the amount of atrophy varied slightly in individual instances the same muscles were affected in all. These were the long flexors of the fingers slightly, the interossei muscles considerably and the muscles of the thenar and hypothenar eminences in varying degrees.

He quotes Herringham as maintaining that the first thoracic nerve sometimes supplies in part the flexores profundus and sublimis, the interossei, the hypothenar and deep thenar muscles.

Buzzard admits that the degree of atrophy which obtained in certain of the hand muscles was much greater than might have been expected if only one out of two or more spinal roots had been cut off, and yet he attributes all the changes he found to lesion of the first thoracic root alone. He had no oculopupillary symptoms. He also found marked anesthesia on the ulnar side of the forearm.

These were all purely clinical cases. He quotes a similar case reported by Charcot.

The case reported by Edwin Bramwell is very similar to those reported by Buzzard, and was without operation or necropsy. It was reported as a lesion of the first dorsal root.

Oppenheim in the 1913 edition of his text-book expresses a doubt as to the correctness of the interpretation of these findings, and the findings both of Buzzard and of Bramwell are considerably more extensive than are those obtained by me in the case in which it was definitely known that only the first thoracic root was affected. I believe that in both Buzzard's and Bramwell's cases the lesion must have implicated more than the first thoracic root.

Many of the diagrams of radicular sensory representation are unsatisfactory for the distribution of the eighth cervical and first thoracic roots. Kocher, as represented by Edinger, makes no attempt to separate these two areas, neither does Seiffer. Henry Head and A. W. Campbell, from their study of herpes zoster, give a distribution for the first thoracic root which conforms remarkably closely with the area of disturbed sensation in my patient, except that the area goes in their diagram all the way to the elbow, which is of minor importance. Dejerine's Fig. 365 in her *Sémiologie*, represents a distribution of the first thoracic root which conforms closely to the area disturbed in my patient, but I can find no justification for the representation she gives of this root as a narrow band in the inner side of the arm extending from the elbow to the axilla. Dejerine does not attempt to separate the motor distribution of the eighth cervical and first thoracic roots.

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ON THE EPIDEMIC ACUTE AND SUBACUTE NON-SUPPURATIVE INFLAMMATIONS OF THE NERVOUS SYSTEM PREVALENT IN THE UNITED STATES IN 1918–1919: ENCEPHALITIS; ENCEPHALOMYELITIS; POLYNEURITIS; AND MENINGO–ENCEPHALO–MYELO–NEURITIS.[1]

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The Clinical Phenomena of the Disease.

This epidemic disease gives rise to clinical phenomena that are referable chiefly to disturbances of the functions of the nervous system. Since any area in the nervous system may be involved, with either loss of, increase of, or perversion of function, and since the single cases vary greatly as to the number and distribution of areas diseased as well as to the intensity of the process in particular areas, we may, in epidemic encephalitis, meet with the greatest variety of neurological syndromes, so that it is not surprising that some clinicians, overlooking an underlying unity, have thought that several of the syndromes correspond to as many different and independent diseases. Just why the intensity of the process and the distribution of the injuries to the nervous tissues are so variable we do not yet know, but there is good reason to believe that in this epidemic we are dealing with a single infectious agent as a cause, and that this agent attacks the central and peripheral cerebrospinal nervous system and its coverings in a widespread, though, at the same time, in a patchy or disseminated way. Under such conditions a great variety of combinations of general and focal symptoms must be expected in a series of cases. An understanding of the underlying pathological-anatomical process in epidemic encephalitis makes clinical diversity no surprise; what arouses wonder is rather the frequency with which certain associations of symptoms, such as lethargy, with fever and opthalmoplegia, are met with in the epidemic. (See Table I.)

[1] This paper was presented (in abstract) before the Association of American Physicians at its meeting at Atlantic City, in June, 1919, and the abstract will be published in the Transactions of the Association.

TABLE I.—CLINICAL PHENOMENA OBSERVED

Case and date examined, 1919.	N. II. Opticus.	N. III (oculomotorius).				N. IV. Trochlearis	N. V. Trigeminus.	N. VI. Abducens.	N. VII. Facialis
		Ptosis.	Ext. bulbar muscles.	Iris.	Accommodation.				
I. C. M. C. Feb. 28	0	Partial bilateral	All weak	Sluggish; anisocoria	....	....	Partial paralysis and sensitiveness of rt. masseter; deflection of jaw to rt.	Paresis of right M. rect. lat.	Bilateral facial paralysis
II. K. S. C. Feb. 28	0	0	0	Sluggish pupils; anisocoria	Difficulty reading	0	0	0	Bilateral paresis and mask
III. N. M. L. Mar. 2	0	0	0	Sluggish; pupils sl. irregular	....	0	0	0	0
IV. K. L. Mar. 13	0	0	0	Miosis and slight irregularity	....	0	0	0	0
V. F. I. Mar. 17		Partial bilateral	Weakness, especially of left M. rect. med.; diplopia	0	Inability to read	0	Sensitiveness of masseters (Thomas's sign).	0	Tremor of upper lips; loss of expressive movements with bilateral paresis of face muscles
VI. D. O. H. Mar. 24	0	0	0	Slight anisocoria and sluggish rt. pupil	....	0	0	0	0
VII. F. X. Mar 22	0	Slight right	Diplopia; weakness of all mm. especially superior rect.	Sluggish pupils	Inability to read for week before admission	Bilateral paresis	Bilateral dysmasesia; tender masseters on ninth day.	Bilateral paresis	Bilateral paresis of lower right facial
VIII. F. M. T. Apr. 14	0	0	Diplopia	Sluggish pupils	Temporary paresis	0	0	Paresis on left side	0

IN CASES OF EPIDEMIC ENCEPHALITIS.

N. VIII (acusticus). Cochlearis. Vestibularis.	N. IX. Glosso-pharyngeus.	N. X. Vagus.	N. XI. Accesso-rius.	N. XII. Hypoglos-sus.	Other phenomena.
0	0	Pulse-rate 108	0	0	Undernourished; gastric hyperacidity; 30 small mononuclear cells in c. s. f. with ++++ globulin and negative Wassermann.
Tinnitus	0	Pulse-rate 104	0	0	Pain in head and neck; nervousness, emotionalism and weakness; aching pains in legs; insomnia; forgetfulness; irritability; difficulty in walking; mental depression; well-marked Parkinsonian mask; marked drowsiness at onset; rather marked, coarse tremor of head and trunk; finer tremor of fingers; systolic pressure 190, diastolic 120; oral sepsis; slight hyperthyroidism; chronic alcoholism, tabagism and strychninism.
0	0	Pulse-rate 90	0	0	Brudzinski +; Kernig +; Babinski equivocal on left; delirium; history of potatorium; in c. s. f. 133 small mononuclear cells; strongly positive globulin and negative Wassermann; "luetic type" of curve with gold sol. test; alb. and casts in urine with phthalein output of 70; 35 pounds underweight; improvement after lumbar puncture; slight fever.
0	0	Pulse-rate 104	0	0	Reflexes normal; no objective disturbance of sensation, but complaint of persistent numbness and tingling in left arm, left loin and left leg, which came on two days after an attack of influenza, and has progressively increased in intensity; ten days after onset, transitory glycosuria; c. s. f. showed 30 small mononuclear cells, positive globulin and negative Wassermann; moderate obesity and slight arterial hypertension; marked oral sepsis.
0	0	Pulse-rate 112	0	Marked tremor of tongue	Marked Parkinson mask; pronounced apathy and depression; delirium at onset after influenza; c. s. f. showed 7 small mononuclears, positive globulin; negative Wassermann; atypical paretic curve with gold solution test; achylia gastrica; myoma uteri; undernutrition 25 pounds; temperature 99 to 100°.
0	0	0	0	0	Onset with backache, hiccough and girdle pains; later severe root pains in lower extremities and later in chest; slight fever; marked drowsiness for 4 or 5 days; unsteady gait; insomnia; c. s. f. showed increased pressure, 48 small mononuclear cells; globulin +++; Wassermann negative; improvement after lumbar puncture; fluid a week later showed 14 small mononuclear cells and ++++ globulin; positive Oppenheim on right.
0	Slight dysphagia	Pulse-rate 100	0	0	Slight fever; drowsiness; unilateral sweating of face; lacrymation; headache; mask-like face; c. s. f. bloody but negative Wassermann; slight nystagmus.
0	0	Pulse-rate 84	0	0	Stupor at onset; long standing psychasthenic state; marked oral sepsis; anemia; slight fever; undernutrition.

ONSET. This may be sudden or gradual, and with or without prodromata (asthenia, lassitude, headache, vomiting). Some of the patients regard the attack as influenzal; others give a history of having had influenza a few days or a few weeks before; still others assert that they have not had influenza at all. The general encephalic symptoms may, in some cases, be the more marked at onset; in others, focal symptoms, referable to brain or spinal cord, or signs of meningeal irritation, announce the presence of a nervous disease.

GENERAL SYMPTOMS. The most striking general symptom, when it is present, is a disturbance of the general consciousness that manifests itself in a pathological drowsiness, a symptom that led von Economo to call the disease "encephalitis lethargica," and the lay press to dub it "sleeping sickness." All grades of this disturbance (apathy, dulness, somnolence, sopor, stupor, coma) may be met with, either in a single case or in a series, and either at or near the onset or later in its course. Some patients, however, do not manifest the symptom at all. In one patient of my series, who was drowsy for about a week, the tendency was attributed at first, erroneously, to the effects of codein administered for the relief of pain. Milder involvements of the general consciousness (somnolence and sopor), from which the patients can be roused and made to answer questions, seem to have been more common in the epidemic than the severer disturbances (stupor and coma).

Other disturbances of the general consciousness, including mental depression, anxiety and delirium, also frequently occur. A patient may be drowsy in the daytime and complain of insomnia and restlessness at night.

Headache, vertigo, tachycardia and vomiting are common general symptoms. Outspoken choked disk was not present in any of the cases of my series, and, judging from the reports, is not met with, though a slight optic neuritis may occur. Fever may or may not be present at the onset, but is usually evident at some time during the course of the disease. It was slight in most of the cases we have seen, though we were impressed with the persistence of an elevation of temperature of 1° or 2°, extending over a period of from one to three weeks in several of the cases. In some cases there is marked pyrexia, 102° to 105°, lasting for from one to a few days.

FOCAL SYMPTOMS. These are predominantly motor rather than sensory.

Commonest by far are bilateral nuclear and radicular paralyses of the eye muscles (of well-known type), with ptosis and ophthalmoplegia externa et interna (polioencephalitis superior acuta involving N. III, N. IV and N. VI); but pontile and bulbar nuclear and radicular paralyses, with facial paralysis, dysmasesia, dysphagia, dysarthria, etc. (polioencephalitis inferior acuta involving N. V (motor), N. VII, N. IX, etc.), are also common, as are symptoms pointing to paralysis of part of the extrapyramidal motor system

(Parkinsonian mask; loss of the normal facial innervations accompanying the emotions). The lesions that are most frequent as causes of motor focal symptoms in epidemic encephalitis must therefore be located in (1) the midbrain about the aqueductus cerebri, (2) the pons and upper medulla oblongata and (3) the basal ganglia (globus pallidus?).

Less common motor disturbances in epidemic encephalitis, though they may occasionally be encountered, are monoplegias, hemiplegias, diplegias, aphasias, contractures, choreatic and athetotic disturbances of motility and general or circumscribed convulsive seizures.

Focal phenomena of a sensory nature are much less frequent than those of a motor nature above referred to. Nevertheless, sensory symptoms due to focal lesions of the sensory areas (cortex) and paths (corona radiata; lemnisci) are occasionally met with (hemi- or mono-anesthesias; paresthesias; hyperesthesias; algias; more rarely, hemianopsia). In one of our cases, in which there was a meningomyelo-encephalitis, severe root-pains in the trunk and lower extremities were prominent.

Acute ataxias of different types (according to the localization of the lesions) may be exhibited. The commonest form is the cerebellar type, due doubtless to lesions of the cerebellum or its peduncles or of the red nucleus mechanisms.

Meningeal Symptoms. Only in relatively few cases are the clinical signs of an outspoken meningeal irritation present. In one case of our series there was pain in the neck and back, some rigidity of the neck, a positive Brudzinski sign and a positive Kernig sign. von Economo, S. A. K. Wilson and others have reported similar cases. That there is a slight patchy involvement of the leptomeninges in the inflammatory process in many cases has been shown, however, by postmortem examinations and by the increased cell count and the positive globulin reaction in the cerebrospinal fluid.

Cerebrospinal Fluid. This may or may not be under increased pressure. The fluid looks perfectly clear, and no film, as a rule, forms on standing. In some undoubted cases of the disease the cell count has been normal and the globulin test negative. The apparent normality of the fluid on naked-eye examination apparently misled many of the European observers, who made the earlier reports on the disease, to declare that no changes are present in the cerebrospinal fluid. Abnormal fluids have been very constantly encountered in our own cases, though they would have been overlooked because of the apparently normal macroscopic appearances had the examiner failed to make a cell-count and a globulin test. *In our experience, a cell-count in the cerebrospinal fluid of from* 10 *to* 100 *small mononuclears along with a positive globulin reaction with negative Wassermann and negative bacteriological smears and cultures*

is, at the time of an epidemic of encephalitis, strong corroborative evidence of the existence of the disease in a patient in whom the process is for any other reason suspected to exist. (See Table II.)

TABLE II.—CEREBROSPINAL FLUIDS IN EPIDEMIC ENCEPHALITIS.

Case.	Day of disease.	Appearance.	Pressure.	Globulin.	Number and variety of cells.	Wassermann reaction.
I. C. M. C.	19th	Clear, colorless	.....	++++	30	0
II. K. S. C.	106th	Clear, colorless	Increased	++++	11	0
III. N. M. L.	21st	Clear (fairly)	Increased	++++	97	0
	23d	?	?	?	Increased; 98% lymph., 2% leuko.	?
	26th	?	?	++++++	133	0
	75th	Clear	Increased	+	33	0
IV. K. L.	66th	?	Normal	+	30; lymphocytes predom.	0
V. F. I.	51st	25 c.c. cl. fluid	Not increased	+	2; lymphocytes	0
	53d	?	?	Weak; pos.	?	0
	66th	?	?	++	7	0
VI. D. O. H.	29th	Clear, colorless	Much increased	+++	48; sm. monos.	0
	36th	Clear, colorless	Much increased	++++	14; sm. monos.	0
	41st	Clear, colorless	Increased, but not so much as formerly	++++	14	0
VII. F. X.	7th	Bloody	?	?	Bloody.	0
	13th	Bloody	Not increased	?	?	?

Negative findings in the cerebrospinal fluid do not, however, rule out the disease.

Still another point regarding the cerebrospinal fluid is worthy of comment. In more instances than accidental injury to a small bloodvessel will apparently account for, a bloody fluid is obtained on lumbar puncture in patients suspected to be suffering from the disease. This point has been noted also by other observers, and it seems likely that the well-known hemorrhagic tendency in the disease is thus sometimes manifested.

TABLE III.—BLOOD IN EPIDEMIC ENCEPHALITIS.

Case.	Day of disease.	Red blood cells.	Hemoglobin, per cent.	White blood cells.	Polymorphonuclear neutrophiles, per cent.	Polymorphonuclear eosinophiles, per cent.	Polymorphonuclear basophiles, per cent.	Small mononuclears, per cent.	Large mononucleara and transitionals, per cent.	Wassermann.
I. C. M. C.	21st	5,212,000	90	6,200	62.0	2.0	0.4	23.2	12.4	0
II. K. S. C.	105th	5,536,000	95	12,800	87.2	0	0	9.6	3.2	0
III. N. M. L.	19th	4,844,800	90	22,850	90.0	1.0	1.0	7.0	1.0	0
IV. K. L.	60th	5,472,000	90	10,000	76 0	0.4	0.0	17.2	6.4	0
V. F. I.	42d	5,856,000	85	10,000	82.8	0.0	0.0	12.8	4.4	0
	57th	?	90	7,700	?	?	?	?	?	?
VI. D. O. H.	30th	4,800,000	85–90	9,000	?	?	?	?	?	0
VII. F. X.	7th	6,328,000	77	9,120	75.5	1.0	0.0	16.0	7.5	0
	9th	6,512,000	75	9,820	76.0	1.0	1.0	13.0	9.0	
	19th	5,280,000	78	7,800	75.0	0.7	0.0	18.7	5.7	
VIII. F. M. T.	30th	5,408,000	97	17,600	70.4	0.4	0.4	22.8	6.0	0
	62d	3,600,000	70	3,058	72.4	1.2	0.0	18.0	8.4	

THE BLOOD. A slight leukocytosis is usually present. The white cell count in our cases varied between 3058 and 17,600. per c.mm., except in one instance in which the cell count was 22,850. The differential count of the white cells usually, though not always, reveals a slight relative and absolute increase in the polymorphonuclear neutrophile elements (70 to 90 per cent. of the total white count). (See Table III.)

THE URINE. A trace of albumin and a few casts are sometimes demonstrable, doubtless due to a slight toxic degenerative nephropathy such as is met with in most acute infectious processes. Renal function, as far as the phthalein test has been applied, appears to be unimpaired. When severer renal disease, as in chronic alcoholism, accidentally accompanies encephalitis with lethargy, the differential diagnosis may be difficult as in one case of our series in which the delirium and coma were thought, at first, to be due to uremic intoxication.

DURATION OF THE DISEASE. Whether the disease terminate in death or in recovery, the course may be either brief or prolonged. In severe fulminant cases death may occur in a few hours or days. In many instances, both mild and severe, recovery has been rapid, the symptoms lasting from a few days to a month. In the majority of cases, however, the disease is a protracted one, extending over many weeks, and in some instances over several months.

PROGNOSIS. As regards life this is much better than might have been expected, even in cases that at onset present the gravest symptoms. The mortality has been variable in different countries, and seems to have been greatest in Austria and in France. Six out of 11 of the cases reported from Austria by von Economo died; 7 of Netter's 15 cases reported from France were fatal; only 2 of S. A. K. Wilson's 13 cases reported from London were fatal; in our own series it happens that there have been no deaths at all, due to accidental encounter of a mild group of infections. Though just what the mortality rate has been in the United States it is as yet difficult to estimate. Bassoe, Pothier, and Tucker have all reported fatal cases, and the newspapers have given evidence of many deaths all over the country from "sleeping sickness."

As regards residues and sequelæ in the patients who recover, it is, as yet, of course, too early to make any definite statement. We have been impressed, on the one hand, with the very remarkable improvement observable in some instances within a few weeks (disappearance of paralyses; recovery of facial expression and vanishing of the Parkinsonian mask; replacement of depression by normal mood), and, on the other hand, with the persistence in some cases over a long period of the eye-muscle disturbances, the dizziness, the depression and the asthenia.

We must be on the lookout in the coming period, we believe, for sequelæ, especially, as it would seem likely that large numbers of the

milder infections must have gone entirely unrecognized. We have the fear, let us hope that it is not well-founded, that, as a result of the epidemic, we may see an increased incidence, later on, in the conditions hitherto known as "multiple sclerosis," unexplained "mental deterioration," and "idiopathic epilepsy."

Nature of the Disease.

Observers had but little difficulty in determining early in this epidemic that they had to deal with an infectious, or toxi-infectious, process involving predominantly the cerebrospinal nervous system. The clinical phenomena of an epidemic disease (of acute onset with fever) that manifested itself by a disturbance of the general consciousness and by paralyses, especially in the domain of the upper, and sometimes of the lower, cerebral nerves left no doubt on this point; but had there been any doubt it would soon have been dissipated by the necropsy findings, which revealed lesions of an inflammatory, and sometimes of a hemorrhagic, character disseminated throughout the nervous system and its coverings (encephalitis; encephalomyelitis; meningo-encephalomyeloneuritis). The resemblance of the clinical features in single cases to various well-known and less well-known syndromes (Wernicke's "polioencephalitis superior acuta;" Strümpell's "primary acute encephalitis;" Wickman's "encephalitic form of the Heine-Medin disease;" Leichtenstern's "post-influenzal encephalitis;" "acute infectious polyneuritis;" botulism with ophthalmoplegia; the mysterious epidemic disease known as "nona" in 1890; and the epidemic "sleeping sickness" (*Schlafkrankheit*) described after a grippe epidemic in Tübingen by Camerarius as far back as 1718) was quickly detected by one or another of those who had opportunity to study the outbreak. An epidemic of an acute infectious (and inflammatory) disseminated meningo-encephalomyeloneuropathy closely resembling, if not identical with, certain diseases earlier described had, then, presented itself in many different countries of the world for study and for consideration.

Concerning the etiology of the disease, very different opinions have been advanced. Some investigators, especially those who studied in the first two or three areas of what is now seen to be a pandemic distribution of the disease evidently thought that they were dealing with an entirely new malady. Thus, von Economo, who, in Austria, described it as "encephalitis lethargica," Bradford, Bashford and Wilson, who studied a special group of cases in the British Expeditionary Force and designated it "acute infectious polyneuritis," and Netter, who in Paris described it as "encéphalite léthargique épidémique," all seem inclined to regard it as a new disease, though they admit its resemblance to recognized diseases. It was soon seen, however, on analysis of the clinical phenomena

and the pathological-histological findings, that under some one or other form of encephalitis, or of encephalomyelitis, parallel phenomena have already been described in medical literature. Thus most of the phenomena, both clinical and histological, will be found paralleled in the larger collective reviews of acute non-suppurative encephalitis, such as those by Chartier (1907), by Oppenheim and Cassirer (1907) and by Vogt (1912). Most of the cerebral-nerve paralyses will be found duplicated in the cases of encephalitis tabulated by Wilbrand and Saenger (1900). Most, if not all, of the pathological-histological findings appear to be in accord with those described by Friedmann (1890–1898), Bücklers (1892), Flatau (1900), Spielmeyer (1902) and Raymond and Cestan (1904). One of the English writers (S. A. K. Wilson) concludes that "what appears to be new is the occurrence in epidemic form of cases of encephalitis not to all appearance due to the action of a known infective or toxi-infective agent, but presenting clinical symptoms any or all of which have been noted in various kinds of encephalitis with which the neurologist has long been conversant." But is even the occurrence in epidemic form new? Have we not records of one epidemic by Camerarius in 1718, of another by Lepecq de la Clôture in 1763, of at least three mentioned by Ozanann (in his history of epidemic diseases) as occurring in 1745, in 1800 and in 1802, of an epidemic of encephalitis described by Leichtenstern after the great influenza epidemic of 1889–1890, as well as of epidemics of "nona" that occurred at about the same time (reports collected and reviewed by Longuet in 1892)? In England the first cases observed in the present epidemic of encephalitis were thought to be instances of meat-poisoning (botulism). The problem of etiology as at present discussed for the recent epidemic resolves itself into three questions: (1) Is the disease due to the virus that causes influenza? (2) Is the disease due to the virus that causes the Heine-Medin disease or to a modification of that virus? and (3) Is the disease due to a virus *sui generis?*

The possibility that the virus that causes influenza may also be the cause of the encephalitis recently pandemic is well worthy of serious consideration; for we have good reason to believe that encephalitis, like meningitis, like pleuritis, like nephritis and like other inflammations, may at different times be due to entirely different causative agents. Thus it is well-known that non-suppurative encephalitis may occur as a complication of scarlet fever, of measles, of chicken-pox, of whooping-cough, of pneumonia, of anthrax, of malaria, of poliomyelitis and of other infectious diseases, and it is probable that these complicating inflammations of the brain are due to local infection of the nervous system with the specific virus that causes the primary general disease rather than that they are all instances of mixed infections of a unitary virus causing encephalitis and of the specific viruses of the several

infectious diseases mentioned. Now if this view be a correct one it is surely not a wholly improbable supposition to suggest that an epidemic, or a pandemic, of encephalitis occurring in association with or soon after an epidemic, or a pandemic, of influenza may be due to an encephalic localization of the influenzal virus. Indeed, the encephalitis that was described by Leichtenstern, in 1890, is generally referred to in the literature as "influenzal encephalitis," and it may be recalled that Pfühl, and later Nauwerck in fresher material, asserted that they had demonstrated the presence of the *Bacillus influenzæ* of Pfeiffer in the diseased cerebral tissues in "influenzal encephalitis." Though the latter demonstration is at present less convincing than formerly, owing to the reopening of the question of the etiology of influenza itself and to the doubt cast upon Pfeiffer's bacillus as its cause,[1] still the occurrence of encephalitis in epidemic (and in pandemic) distribution in such close connection with epidemic (and pandemic) influenza as that observed in 1890–1892, and as that at the present time, strongly suggests at least one of the three following possibilities: (1) that the influenzal virus is the cause of the epidemic encephalitis, (2) that influenzal infection predisposes to infection with the virus of encephalitis, or (3) that the cosmic influences that determine the occurrence of epidemic influenza also determine the occurrence of epidemic encephalitis, even if each disease is due to a separate and distinct virus. It may be argued also that influenzal encephalitis tends to assume a special clinical form (acute cerebritis), but certainly other forms also occur in the epidemic; indeed, in the present outbreak, the polioencephalitis-superior type has predominated. Granting the weight of the arguments adduced, it must be admitted, however, (1) that the incidence of epidemic encephalitis is very low compared with that of influenza, (2) that the available data do not permit of the assertion of any direct parallelism of incidence and distribution in the two diseases, and (3) that some of the patients who have suffered from encephalitis deny any preceding attack of influenza, though doubtless all have been exposed to that disease. Thus until we are sure of the precise nature of the virus of influenza on the one hand and of the virus of epidemic encephalitis on the other, the questions regarding the exact etiological relationships of the two diseases must be left open.

It is but little wonder, too, that some have thought that the virus of epidemic encephalitis may be identical with, or a slight modification of, the virus (*Flexneria noguchii*) that is the cause of the Heine-Medin disease (poliomyelitis anterior acuta). For, in the first place, since Wickman's studies, cerebral, pontile, cerebellar, bulbar and meningeal forms of the Heine-Medin disease are certainly known to occur. And, in the second place, the pathological-histological lesions in the central nervous system and the findings in the cerebro-

[1] Cf. Bloomfield, A., and Harrop, G., et al.

spinal fluid in epidemic encephalitis and in poliomyelitis show a close resemblance. But against the view of identity there are many facts that may be urged. The onset of the paralyses is very different in the two diseases, since in poliomyelitis the paralyses are all there from the beginning, whereas in epidemic encephalitis the paralyses begin insidiously, often as ptosis or loss of power of accommodation, and gradually extend through a period of several days or longer to other muscles. Bilateral symmetry of the paralyses is more common in encephalitis, unilateral or asymmetrical paralyses in the Heine-Medin disease. The fever is usually slight and often more marked after the paralyses have developed in encephalitis; in the Heine-Medin disease the fever is, as a rule, highest before paralysis occurs. There is a much higher leukocytosis in the blood and also a higher cell count in the cerebrospinal fluid in the Heine-Medin disease than in encephalitis. The age-incidence is also different, since encephalitis is common at all ages of life, whereas the Heine-Medin disease shows a special predilection for childhood and adolescence. Again, a careful study of the distribution of the cases of encephalitis and of poliomyelitis in England has shown that the two series of cases are distributed independently of one another. And, finally, if there were identity of etiology one would expect more typical encephalitis cases in epidemics of poliomyelitis and more typical poliomyelitis cases in epidemics of encephalitis than we actually see. Some investigators, though fully granting that there may not be identity of etiology, suggest that the virus of encephalitis may be one special strain and the virus of poliomyelitis another special strain of a single microörganism, just as we now recognize Type I, Type II, and Type III, etc., of the *Pneumococcus*, or recognize that *Bacillus paratyphosus A* and *Bacillus paratyphosus B* are two strains producing similar but not identical diseases. In favor of this view may be adduced the findings of von Wiesener, of Vienna, who asserts that he has isolated an organism (globoid diplococcal form) from encephalitis and reproduced the disease in monkeys, and that this virus resembles closely that isolated by Flexner and Noguchi in poliomyelitis. A similar virus seems to have been grown by Wilson from Bradford's "acute infectious polyneuritis" cases, and with this virus it was also possible to reproduce in monkeys what they believed to be the disease they had under study. Cleland and Campbell assert that they have successfully conveyed the virus of the disease to the sheep, the calf and the horse. It is, of course, possible that just as we have a group of diseases (the leishmaniases) due to different strains of species of *Leishmania*, so we may have a group of diseases (the flexneriases) due to different strains or species of an organism (*Flexneria*), of which the virus of the Heine-Medin disease is one. But until more extensive virus studies and experiments have been made it would be unwise to look upon such a view as anything more than an interesting speculation.

That epidemic encephalitis may, finally, be due to some causative agent neither identical with nor even a modification of any other virus is quite possible, and is, indeed, the view held by a number of those who have studied the disease. If this view turn out to be the correct one it will be interesting, later on, to find out whether the virus isolated by von Wiesener, that isolated by Wilson, or some entirely different filtrable (or unfiltrable) virus is the etiological agent.

Treatment of Epidemic Encephalitis.

The patients under our observation have evidently been of the milder type, as none of them died. Convalescence has been tedious in several of them, and in how far there will be residual phenomena in some, it is as yet too early to be sure.

At the onset of the disease, rest in bed, protection from external stimuli of all kinds, laxatives, bland diet and relief of headache and of pains would seem to be desirable.

In our experience, lumbar puncture, done for diagnostic reasons, relieved the symptoms so markedly in several instances that it was repeated at intervals as a therapeutic measure.

During convalescence, prolonged rest, careful nursing, a nutritious diet and mild hydrotherapy, electrotherapy and massage have been the only measures made use of. Complete recovery without residuals seems to be common.

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PLEURAL EFFUSION WITH INVERSION OF THE DIAPHRAGM PRODUCING AN ABDOMINAL TUMOR; TOGETHER WITH REMARKS ON ACUTE PULMONARY EDEMA FOLLOWING TAPPING.[1]

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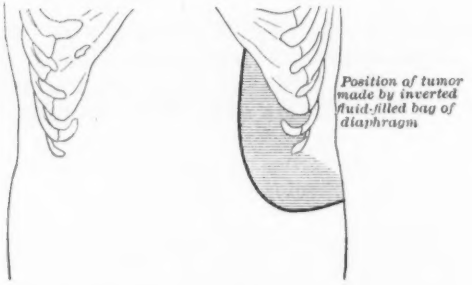
PHILADELPHIA.

A NUMBER of years ago I made an autopsy at the Philadelphia General Hospital on a case of left-sided pleural effusion. On opening the abdomen I found in the left upper quadrant a large, smooth, tense tumor which upon further exploration proved to be a bag made by the inverted diaphragm and filled with pleural fluid. The incident made a deep impression upon me, but was not duplicated in my further experience as pathologist. I have, however, seen this tumor twice since then, clinically. Of the first case I cannot find any record; the second I saw a few months ago; it is as follows: The patient is a widow, aged seventy-seven years, who came under my observation in April, 1918. The salient points in her case are as follows: For years she has suffered with diabetes mellitus and chronic nephritis with moderate hypertension. She has diabetic cataract and retinal hemorrhages. When I first saw her, the heart was slightly enlarged; there was a soft systolic mitral murmur; the legs were somewhat edematous. The urine contained sugar up to 3.2 per cent.; and albumin from traces to 2.8 grams per liter. Ketones were inconstantly present. The quantity of urine

[1] Read before the Section on Medicine of the College of Physicians of Philadelphia, October 27, 1919.

was usually about 1500 c.c.; specific gravity varied from 1007 to 1013. Unlike most cases in which nephritis and diabetes coexist, the sugar in this patient did not vary in inverse proportion to the albumin—the larger amounts of sugar were often found when the albumin content of the urine was high.

Under treatment the patient improved greatly and was able to spend the summer of 1918 very comfortably in Atlantic City. On July 25, 1919, I saw her at her home in Wilkes-Barre with Dr. Bullard. It appeared that after eating some cheese on June 5, she had been seized with an attack of acute indigestion characterized by abdominal pain radiating to the back, nausea, vomiting, and slight diarrhea. Within a few days thereafter she began to suffer from shortness of breath and fluttering of the heart which kept her confined to her room. I found her sitting up, intensely short of breath; the lips somewhat cyanosed; pulse rapid; no fever. Making a cursory examination of the abdomen I detected on the left side a large, tense, rounded mass, slightly uneven, somewhat tender, and



occupying nearly the entire upper left half of the abdominal cavity. It extended forward to the prolonged midclavicular line and downward to the level of the umbilicus (see figure). I was at first somewhat nonplussed by it, but found the explanation when I examined the chest. The left chest was visibly distended and enlarged. The percussion note was flat from above the left clavicle down to the bottom of the thorax, front and back. No heart sounds could be heard on the left side; the apex beat was distinct both on palpation and on auscultation in the fifth interspace just within the right nipple line. Over the left chest bronchial breathing and a twangy bronchophony could be heard. I concluded that the abdominal tumor was the inverted diaphragm containing pleural fluid, a view confirmed by the results of tapping the chest.

After drawing off the fluid, which was done slowly, five pints being obtained, the tumor could no longer be felt. Fearing the possibility of pulmonary edema on account of the presumably long duration of the effusion, I remained for a considerable time at the

patient's bedside. In the first half hour nothing happened; the patient was comfortable and greatly relieved of her shortness of breath. Then suddenly the lungs filled up with fluid, loud bubbling rales could be heard everywhere throughout the chest; the breathing became rattling and terribly labored. A violent cough which brought up only a little fluid added to the distress of the patient whose air hunger was painful to see. Her face became purple; the skin cold and clammy; dissolution seemed imminent. I gave her a hypodermic injection of morphin and atropin, which in other cases of this type I had seen productive of good results, but it had no effect. I then applied a number of dry cups over the back, and as if by magic every rale disappeared. I have never seen anything quite so dramatic. The grandson who was present in the room and who had been a most anxious spectator was amazed beyond expression by the instantaneous improvement.

After a proper interval I removed the cups; there was an immediate return of edema, but it was slight and caused little distress. From that time on the patient's improvement was rapid, and she was able to go to Atlantic City the latter part of July, where I had the pleasure of seeing her with Dr. William W. Fox. A considerable accumulation of fluid had again taken place and the abdominal tumor had reappeared, but was not as large as before. Sometime later Dr. Fox tapped the chest, the tumor vanished, and the patient was relieved of her distress. As I know from frequent reports she is in good condition to this day.

I have looked through many text-books on diseases of the lungs and through recent and old works on physical diagnosis without finding more than two references to the diaphragmatic inversion.

Wilson Fox in his monumental work,[2] mentions it in the following words: "When the effusion is large, the diaphragm may pass below the ribs and may there, in some cases, be felt as a tumor."

Collet[3] describes the condition clearly, although he does not bring out the fact that a real tumor-like mass may appear in the left upper abdomen. He says: "Dans les epanchements énormes qui écrasent le diaphragme au point de le faire bomber dans l'abdomen, la matité franchit les limites de l'aire de Traube et dépasse de plusieurs travers de doigt le rebord costal."

The condition is a mechanical result of great intrapleural pressure. As pointed out by Powell and Hartley[4] "the negative pressure within the pleura is maintained until the lung is completely retracted before the advancing fluid; thereafter any further accumulation compresses the lung and by its weight and pressure forces down the diaphragm." To invert the diaphragm, however, to turn it inside out, a much greater pressure than ordinarily exists in pleural

[2] Diseases of the Lungs and Pleura, London, 1891, p. 983.
[3] Précis des mal. de l'appareil respir., Paris, 1914, p. 501.
[4] Diseases of the Lungs, London, 1911, p. 97.

effusions must be produced. If the patient is up and about, gravity probably acts as a favoring factor.

It is evident that a tumor formed by a bag of pleural fluid projecting into the abdomen must be considered in the differential diagnosis of abdominal masses. If the chest condition is overlooked, or when not overlooked, if the pleural effusion and the abdominal tumor are not correlated, the possible diagnostic errors are many. The tumor has a peculiar bulky feel, is tender to touch, does not move with respiration, has the shape neither of the spleen nor of the kidney, and appears to have a deep attachment. Its true nature is revealed after aspiration of the chest, when it will be found to disappear. In the absence of tapping a roentgenologic examination is probably the best means of diagnosing the true nature of the tumor. Such an examination will likewise show whether after tapping the diaphragm has returned to its normal position and is functioning properly.

Several years ago in a paper on "Albuminous Expectoration Following Thoracocentesis,"[5] I referred to acute pulmonary edema following tapping, ascribing it to "congestion by recoil." It is very alarming and sometimes terminates fatally. Usually a hypodermic injection of morphin and atropin brings speedy relief, but, as in the case just cited, it may fail. The remarkable results produced by dry cupping, which cannot be questioned, must be due to some reflex nervous influence. I believe dry cupping to be a good therapeutic measure in all forms of acute pulmonary edema whether due to tapping or to other causes.

RETROPERITONEAL LIPOSARCOMA: REPORT OF AN UNUSUALLY LARGE SPECIMEN, WITH CHEMICAL ANALYSIS.

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and

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Some time ago one of us made an investigation of the reputed ability of lipomas to retain or even to lay on fat when the body of the host is losing fat, perhaps to the extent of extreme emaciation.[1] The positive evidence recorded in the literature was found to be very scanty, and there were instances in which the lipomas had decreased in size when the host was losing weight. Analyses of lipoma fat have not shown any detectable difference from the fat

[5] Am. Jour. Med. Sc., April, 1902.

[1] Wells: The Fat Metabolism of Lipomas, Arch. Int. Med., 1912, x, 297.

of normal fatty tissues to explain why it cannot be utilized for the nutrition of the body, and experiments with the fat-splitting enzymes of lipomas did not indicate any significant difference from the enzymes of fatty areolar tissue that could be detected by the crude methods available. The literature does establish, however, that malignant fatty tumors continue to grow while the host is becoming emaciated.[2] This is especially true of the malignant retroperitoneal fatty tumors, which are so often of very great size, constituting, as a class, probably the largest of the solid tumors.

These retroperitoneal tumors of fibrous and fatty tissue, developing slowly and recurring occasionally after removal, are reported fairly often in the literature, some of them having attained large dimensions. Their peculiar interest seems to rest largely with this slow expansile growth and the unusual size that some have attained. Because of their structure these tumors have received various designations, such as myxoma lipomatodes, lipoma myxomatodes, lipoma edematosum myxomatodes, myxoma undergoing fatty degeneration, myxolipoma, myxo-fibro-lipoma, myxo-sarco-lipoma, etc. Without doubt the tumors described under these various designations could be grouped under one general name, for each contains fibrous and fatty tissues in different proportions, usually with more or less malignant degeneration. Robertson,[3] in his review of the literature, records the frequency of these retroperitoneal tumors, gives the gross and the microscopic characters, and includes other special features, such as the weight where this has been mentioned. He collected 51 recorded cases of tumors of this histological type, adding one of his own. Of these, 17 were retroperitoneal and 3 in the mesentery, most of the others arising in the arm or leg.

Gardner and Adami,[4] in 1900, reporting the occurrence of tumors of this kind, stated that Waldeyer up to that time had recorded the largest. This neoplasm weighed sixty-three pounds, had developed in the mesentery and had formed secondary metastases in the liver and lungs. McConnell[5] in 1908 described a tumor arising in the right kidney region, which he considered a hypernephroma that had undergone fatty changes accompanied by a limited tendency toward malignancy. The description given is, however, not adequate to decide the point of origin of the growth, and its general features suggest its origin in the perirenal fatty tissues. The entire tumor removed surgically weighed sixty-five pounds, and at a subsequent operation, six and a half years later, a recurrent growth

[2] Adami: Montreal Med. Jour., 1897, xxv, 529. Voeckler: Deut. Ztschr. f. Chir., 1908, xcix, 149.

[3] Lipoma Myxomatodes, Jour. Med. Res., 1916, xxxv, 130–146.

[4] On a Case of Retroperitoneal Lipoma (Lipoma Myxomatodes) with Accompanying Retroperitoneal Fibroma (Chondromyxofibroma), Montreal Med. Jour., 1900, xxix, 417–421.

[5] Recurrent Liposarcoma of the Kidneys, Jour. Med. Res., 1908, xix, 225.

weighing twenty-seven pounds was removed. McConnell's tumor to date is the largest of its kind recorded in the literature.

We have had the opportunity to study a typical example of a fatty retroperitoneal tumor of the classical type, which happens to exceed in size the largest tumors previously reported and which illustrates exceedingly well the parasitic capacity of tumor tissue to hold fat needed by a greatly emaciated host. In addition a chemical analysis of the tissue has been made which throws some light on the character of the tumor.

CASE HISTORY.—The bearer of this tumor was a white male, aged fifty-five years at the time of his death in the Cook County Hospital, March 1, 1917, The clinical history is briefly as follows:

The patient entered the hospital the first time April 4, 1915, and complained of shortness of breath, with moderate cough, swelling of abdomen and feet, loss of appetite and weakness. He stated that the trouble started eighteen months before. At that time he noticed a swelling of the abdomen and later the feet also became swollen. Swelling of the feet and shortness of breath have recurred several times since, the last attack occurring ten days before entering the hospital. These last attacks were much more severe and were accompanied by slight pains in the left iliac fossa. Swelling of the abdomen has been gradual. Appetite has been poor for several weeks. He has had no gastric pain, but occasionally has a sense of fulness, which is sometimes accompanied by vomiting after coughing spells. This condition has no relation to meals or to food eaten. He has had difficulty in starting urine, and dribbling occurs at the end of urination. He had had no previous illness until two years ago, when he was operated on for inguinal hernia, and a tumor was removed in the lower part of the abdomen. He had gonorrhea and chancre twenty-six years ago. He drinks two or three whiskies a day. Even at that time, two years before his death, emaciation was very marked. The blood count was 5,100,000 reds and 10,300 whites.

On examination the abdomen showed marked distention and the veins on the lateral wall of the abdomen were greatly distended. Nodular masses were present in the abdomen, especially in the epigastrium. Dulness was distributed irregularly over the entire abdomen. There was dulness in the flanks and an umbilical hernia was present. Paracentesis yielded no fluid on the first attempt, but a second trial gave 5 c.c. of fluid, which contained large lymphoid cells and many eosinophiles showing peculiar mitotic figures. A Wassermann test of the blood was negative. A diagnosis of carcinomatosis of the peritoneum was made and the patient left the hospital April 12, 1915.

He returned to the hospital September 8, 1915, with the same complaints as before. Previous history was verified and examination showed practically the same condition as before. He had gained

nineteen pounds, but the face and neck were still greatly emaciated. He had passed bright red blood from the rectum lately, but had no pain on defecation. Upon lying down he did not show the dyspnea which a cardiac case with the same amount of dyspnea would show. The heart and lungs were both compressed. The liver was large and hard. Hemorrhoids were present. A large amount of ascites was described, but on paracentesis only 40 to 50 c.c. of clear pale yellow fluid was obtained. A diagnosis of inoperable carcinoma of the peritoneum was made and the patient was discharged September 18, 1915.

FIG. 1.—Photograph of patient a few days before death, showing distention of abdomen produced by retroperitoneal tumor.

He reëntered the hospital February 23, 1917, and his appearance at this time is shown by the photographs. In addition to his former complaints he stated that he had difficulty in urination and defecation. He urinates every twenty minutes, and very small amounts each time. He now drinks five or six whiskies a day. Paracentesis

yielded 50 c.c. fluid, which was slightly blood-tinged. The Wassermann test was negative. The blood showed 12,800 leukocytes. Urine contained fine granular and coarse granular hyaline casts, a few red blood cells and a few epithelial cells. The fact that the patient had lived nearly two years after the first diagnosis of inoperable carcinoma of the peritoneum was made rendered this diagnosis most improbable, and it was thought that there must be a cystic condition, presumably loculated, because of the small quantities of fluid obtained by puncture. Therefore, laparotomy was performed by Dr. Kellogg Speed February 27, 1917.

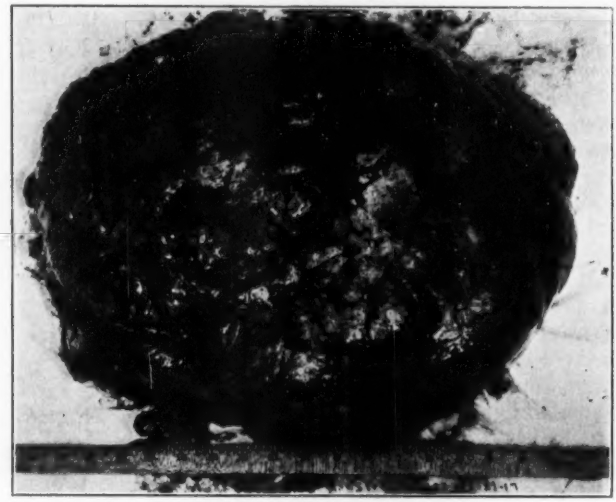

FIG. 2.—Tumor mass removed at operation.

A large tumor mass was found between the anterior two layers of the mesentery. It was approximately the size of five adult heads, and the surface was smooth, irregular, glistening and of medium consistency. Fig. 2 shows the gross appearance of the tumor mass removed at operation. It was bound to the mesentery by tough adhesions. A large kidney-shaped mass could be seen on the right side, and between the posterior two layers of mesentery two other tumor masses could be palpated. One was firmly bound down by adhesions and seemed to be attached to the spleen, which was also enlarged. The liver was easily palpated, but was exceedingly small and shrunken. The stomach could not be located. Most of the intestines were found in the lower quadrant of the abdomen. In one part of the transverse colon a small puncture wound was caused by tearing off an adhesion. The tumor mass between the anterior two layers of mesentery was removed and the wound closed. This

mass, which weighed 14,340 grams, is described in the autopsy protocol. The remaining portion of the tumor could not be removed. Following the operation the patient remained in a collapsed condition and died after about forty-eight hours, on March 1.

The appearance of the patient a few days before the operation is well shown in the accompanying photograph.

AUTOPSY FINDINGS. The autopsy was performed a few hours after death and the significant features are given below.

External Appearance. The body is that of an extremely emaciated man, 162 cm. tall. The legs are slightly edematous, the left more than the right, and the abdomen is greatly distended. The face, arms and chest are greatly emaciated. The umbilicus is very prominent, and a hugh recent incision, 30 cm. in length, extends along the midline of the abdomen, closed with catgut and skin clips. A large mass can be palpated in either flank. The external genitals are very edematous. There is a nodular mass, 9 cm. by 6 cm., in the right inguinal canal, and there is the scar of an operative wound over the right inguinal canal. The scrotum is stretched. There are superficial ulcers on the right leg. The veins of the left leg are distended. There is a sutured incision in the bend of the right elbow. The present circumference of the abdomen is 100 cm.

Abdominal Cavity. There is extremely little subcutaneous fat. The subcutaneous tissues in the abdominal wall are edematous and hemorrhagic. The fibrous tissue in the midline is distinctly hypertrophied. There is about a liter of fluid, somewhat blood-stained, in the peritoneal cavity. A tumor, recently removed by surgical operation from the abdominal cavity, was also examined. It is nodular, with adhesions of highly edematous connective tissue, very soft, jelly-like, pale and semisolid. The cut surface is gelatinous and watery, divided into lobules 5 to 10 cm. in diameter, but no definite cyst formation is present. This tumor mass weighs 14,340 grams.

A sutured incision in the peritoneum, about 15 cm. long, indicates the site of the pedicle of the removed tumor mass, at the root of the mesentery, where it grew forward from the mass of similar retroperitoneal tumor tissue.

The abdomen is still filled with the same kind of tumor tissue, covered with peritoneum. It arises in the root of the mesentery and is smooth and movable, and there is no inflammation of the peritoneum.

There are fresh adhesions along the point where the tumor was removed. The upper portion of the tumor is softer than the lower portion. A portion of the tumor mass extends under each psoas muscle; these masses each measure about 7 x 4 x 5 cm. A tumor mass continuous with the large mass extends down into the right inguinal canal. This entire tumor mass is very similar in structure and composition to the one that had been removed. It measured,

on removal, 45 x 20 x 15 cm., and weighed 17,300 grams. One small area of this tumor is pinkish and fleshier than the remainder. The left inguinal ring admits the tip of the finger for a distance of 2 cm. The right inguinal canal containing the tumor mass is very large. The appendix is stretched out and elongated by the tumor growth, to which it adheres; it is 15 cm. long. The tumor fills the entire retroperitoneal tissues from the dome of the diaphragm, which it pushes up, down into the pelvis. Everywhere it is covered by a smooth fibrous capsule and shows no tendency to infiltrate. Everywhere it is pale, often watery and semifluctuating, but nowhere cystic or hemorrhagic. It yields no slimy or mucinous material. There are areas of lobulated fatty tissue passing directly into the edematous fibrous tumor tissue; also, there are areas where the tissue is more fleshy and slightly pinkish. The several types of tissue seem to be separated into distinct lobules, generally from 4 to 10 cm. in diameter. The right kidney and adrenal are embedded in the tumor mass, but not adherent; left kidney and adrenal not embedded. The mesentery is invaded slightly, but still leaves a free mesentery about one-half the normal length. Intestines free and nowhere adherent. There is only a little of the tumor tissue in the pelvis, although it follows the external iliacs along the brim of the pelvis and passes through the right inguinal canal. The pancreas is free from the tumor. Omentum not involved and lesser peritoneal cavity is empty and free from adhesions.

Pleural Cavity. Both pleural cavities are free from adhesions and contain very little fluid.

Pericardial Cavity. The pericardial cavity contains a small amount of clear fluid. The pericardium is smooth.

Heart. The heart is normal and weighs 300 grams. The coronaries are thick-walled and tortuous. The aorta is free from sclerosis and is not surrounded by the tumor mass, which lies mostly anterior to it. The pulmonary arteries are normal. Both iliac arteries are surrounded by tumor masses. The vena cava is somewhat compressed, but there are no thrombi. Just above the bifurcation the sides of the vessel are compressed and adhesions of apposed intima have formed, completely occluding the left common iliac vein, but it contains no thrombi. The portal vein contains no thrombi.

Lungs. The lungs are small and collapsed. The right lung weighs 500 grams and there are no nodules in the lung or pleura. The lower lobe is boggy and semisolid. The left lung is more boggy than the right and weighs 850 grams. It is full of turbid fluid except the anterior margin of the upper lobe. The cut surface shows grayish areas, with central softening and gray consolidated areas. The pleura above these areas is not roughened.

Peribronchial Lymph Glands. The peribronchial lymph glands are very anthracotic, but not greatly enlarged. One on the right side shows a gray caseous area.

Liver. The liver is crowded up with the diaphragm, the dome of which reaches the lower border of the first rib on the right side and the lower border of the second rib on the left side. The liver has no relation to the tumor except for adhesions about the right lobe and the peritoneum over the tumor. The liver weighs 1800 grams. The right lobe is increased slightly and the left lobe is atrophied in the outer half, where it is reduced to a layer 3 to 7 mm. in thickness. The gall-bladder is slightly enlarged. The common bile duct is not distended. The cut surface of the right lobe is of a grayish color. There is a well-defined solid area in the middle part of the right lobe, wedge-shaped, 6 cm. wide and 6 cm. deep. It is dark in color and shows autolysis, but there are no thrombosed vessels in this area. The cut surface of the left lobe shows loss of hepatic tissue, with connective tissue remaining.

The spleen, adrenals and pancreas appear normal.

Gastro-intestinal Tract. The stomach is normal. The descending colon and sigmoid are about twice the normal length and stretched about the tumor. There are dense adhesions between the colon and the tumor, and there is great edema in the vicinity of the hepatic flexure. There is great induration of the fibrous tissue about the cecum. The appendix is greatly elongated by a tumor mass between it and the cecum.

Kidneys. The kidneys weigh 300 grams. Both are firm and pale and the cortical markings in both are obscure. The veins about the left ureter are greatly distended, but the ureter is not distended. The right kidney is deeply embedded in tumor tissue, which does not infiltrate or adhere. The capsule on each strips easily, leaving a smooth surface. The ureters and urinary bladder appear normal.

Generative Organs. The right testicle is soft, large and flattened. There is a marked atrophy of the glandular tissue, but there is no change in the epididymis. The left testicle has increased fluid in the sac. It is large and the glandular tissue is normal. The right seminal vesicle contains fluid, but the left seminal vesicle is empty. The prostate appears normal.

The lymph glands of the abdomen show no enlargement or other abnormality.

Muscular System. There is a marked diminution of muscular tissue throughout the body.

Skeleton. The vertebral column is normal and not involved by the tumor growth. The ribs flare out at the base.

Anatomical Diagnosis. Retroperitoneal, edematous fibro-liposarcoma; slight ascites; compression of both lungs, with hypostatic edema of right lower lobe and left upper lobe; acute hypostatic and aspiration pneumonia of the left lower lobe; upward dislocation of heart; pressure atrophy of the left lobe of liver; occlusion of the left common iliac vein by compression and adhesion; edema and varicose veins of both lower extremities; early varicose ulcers of the right

leg; chronic edema of the external genitals; tumor mass in the right inguinal canal; fibrosis of the right testicle; recent laparotomy wound; fibrocaseous right peribronchial lymph gland; enlargement of the chest cavity; recent phlebotomy wound of the right elbow; emaciation.

Histological Examination. The tissues outside the tumor showed nothing of particular interest, corresponding in all respects to the gross findings. No signs of metastatic growth or tumor cell emboli were observed. The chief findings in the tumor itself were as follows: Histologically the tumor shows the same degree of variation that

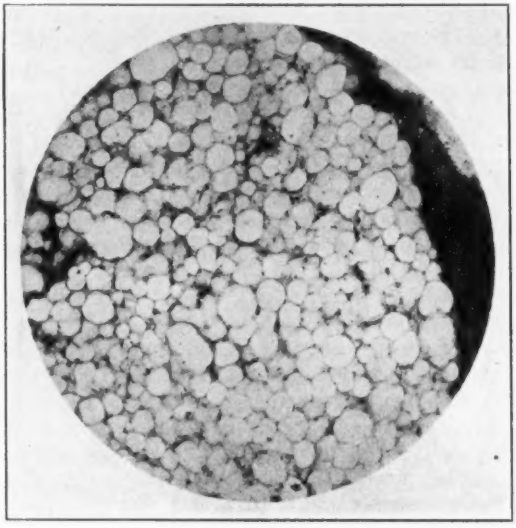

FIG. 3.—Fatty portions of tumor, showing fat cells greatly distended with fat, in contrast with the completely exhausted condition of the normal fat deposits shown in Fig. 6. In the stroma elements irregular and abnormally large nuclei will be seen. × 60.

it does to the naked eye. There are areas that seem to be composed of pure fatty areolar tissue, but even in the parts that most closely resemble a benign lipoma there is occasionally found in the interstitial tissue a large deeply staining and altogether abnormal nucleus (Fig. 3). This is also true of the well-defined fibrous capsule that surrounds all parts of the growth, even in the areas where it is covering the most typical fat tissue. The more solid fleshy portions of growth consist of a structure that in most respects resembles an ordinary fibrosarcoma, but always more or less atypical (Fig. 4). The edema of the intercellular substance and the tendency for the appearance of multinucleated cells and atypical cells, with

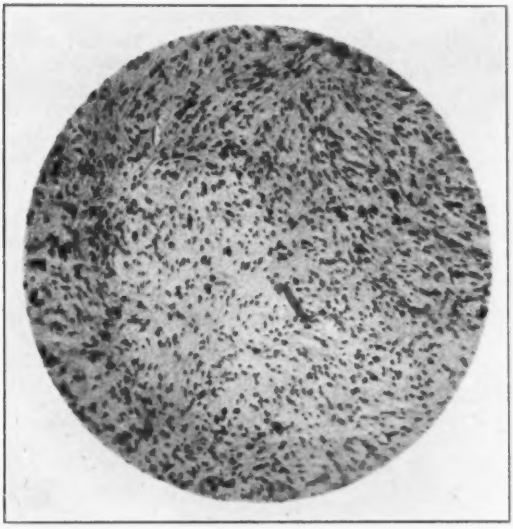

FIG. 4.—Fibrosarcomatous tissue which makes up the bulk of the tumor. Note the numerous exceptionally large and irregular nuclei. × 110.

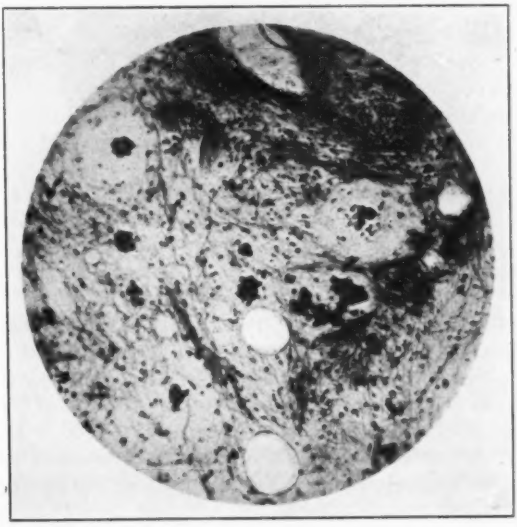

FIG. 5.—Showing the edematous tumor tissue, and the multinucleated cells which are conspicuous in this growth. × 110.

single, large, irregular and often bizarre nuclei, are marked features (Fig. 5). All possible transitions between the fat tissue and the solid fibrosarcoma are seen. A large proportion of the tumor consists of fat cells, with excessive amounts of fibrous tissue containing varying numbers of abnormal cells.

It is to be noted that the fat cells are almost always fully distended with fat, and do not present at all the appearance of fat tissue that has given up its fat for metabolism. This is in marked contrast with the structure of the normal fatty areolar tissues elsewhere in the body, which show almost complete disappearance of the fat deposit, to about as extreme a degree as is possible, as shown in Fig. 6.

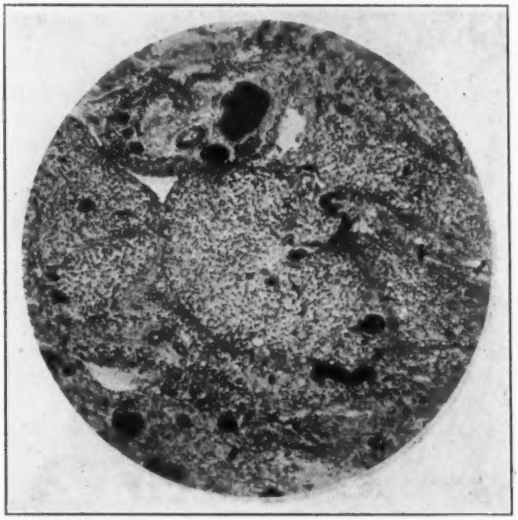

FIG. 6.—Fatty areolar tissue about the adrenal. Illustrating the completely exhausted state of the fat cells, which contain practically no fat whatever. Compare with Fig. 3, showing the fatty tissue of the tumor magnified to the same degree. × 60.

The giant cells are extremely varied in their structure (Fig. 5). The commonest form exhibits a small cluster of deeply staining nuclei, ten to twenty of which appear in a single plane. Often these nuclei appear at the periphery, resembling somewhat a Langhans giant cell, but differing essentially in the exaggerated size of the nuclei, their intense staining and the relatively large proportion of the giant cell that they form. In some, however, the nuclei of the giant cells are in the center, often with a greatly vesiculated cytoplasm. Mitotic figures are not seen, but many nuclei give

the impression of direct division. In some areas numerous small round cells are present and also many cells with a small dark nucleus and nearly homogeneous eosinophile cytoplasm.

The collagen fibers are well developed and made conspicuous by the edema of the tissue. Bloodvessels are usually well developed and in places are extremely numerous, although, for the most part, the degree of vascularity is not different from that of ordinary fat tissue. There are but few polynuclear leukocytes. There are considerable areas of the tumor where very few nuclei are seen although large amounts of stroma substance remain. In the vicinity of these areas there are usually few of the large atypical cells, and the impression is created of a degeneration of the tumor tissue from the lower blood supply. Nowhere is there found any material resembling mucin in its basophilic properties. While sometimes cells with branching processes occur, they do not have the typical mucoid matrix about them, and hence they resemble the stellate cells commonly seen in edematous fibrous tissues. These microscopic observations agree with the absence of any appreciable quantities of material behaving in solubility like mucin.

It is to be remarked that in this tumor we have found no evidence of mucin or so-called myxomatous tissue. The growth is essentially a highly edematous fibro-lipo-sarcoma.

The microscopic difference between this tumor and tumors properly designated as lipomamyxomatodes is very distinct, and emphasized by comparison with sections from a tumor of the latter type in our collection. This specimen was brought to the laboratory many years ago, with the statement that it was a lipoma which had been removed from the right Scarpa's triangle of a woman, aged forty years. It had been present for three years and had shown rapid growth only in the two months preceding the operation. The growth had a distinct capsule and was readily shelled out. It resembled an ordinary lipoma in being lobulated and fatty, but was much softer than a lipoma, very watery, with a slimy fluid escaping from the cut surface, which was extremely pale, with a boiled-starch appearance. It was nearly spherical, approximately 15 x 13 x 10 cm.

Microscopically the tissue everywhere consists of an intimate mingling of fat cells and myxoma cells, with an abundant mucoid deposit possessing marked basophilic properties. There is an absence of edematous connective tissue or of giant cells and other sarcoma cell types. However we were informed that there was rapid recurrence after the operation with early death.

Chemical Composition. Samples were taken from different parts of the tumor tissue removed at autopsy, as nearly as possible securing a fair average of the tumor as a whole, and these samples were united and analyzed.

Extraction with $Ca(OH)_2$ yielded but a trace of material precipi-

tating on acidification with acetic acid, indicating the absence of any considerable quantity of mucin.

The figures obtained are given in the following summary, together with the figures obtained by analyzing a typical old granulation tissue, as existing in the so-called "castration tumors" in swine. These great granulomas are quite common, apparently resulting from a chronic infection of the operation wounds. Histologically they consist for the most part of dense fibrous tissue, with few cells and a scanty blood supply.

	Fibro-liposarcoma.	Granuloma.
Alcohol ether residue	6.53 per cent.	15.84 per cent.
Alcohol ether extract (dried)	2.94 "	2.28 "
Total solids	9.47 "	18.12 "
Water	90.53 "	81.88 "

ALCOHOL ETHER RESIDUE.

Total protein	669.1 mgm. per gm. solids	911.6	mgm.
Protein sulphur	6.5 mgm "	3.3	mgm.
Protein phosphorus	4.4 mgm. "	3.1	mgm.
Total purin nitrogen	1.3 mgm. "	0.7	mgm.

ALCOHOL ETHER SOLUBLE.

(*a*) EXTRACTIVES.

Total inorganic (ash)	61.3 mgm. per gm. solids	19.2	mgm.
Total solids	129.2 mgm. "	66.4	mgm.
Total sulphur	1.2 mgm. "	0.5	mgm.
Inorganic sulphur	0.3 mgm. "	trace	
Total phosphorus	1.5 mgm. "	0.9	mgm.
Inorganic phosphorus	1.3 mgm. "	0.7	mgm.
Total nitrogen	7.9 mgm. "	3.4	mgm.
	49.0 mgm. protein	21.9	mgm. protein.

(*b*) LIPINS.

Total nitrogen	1.7 mgm. per gm. solids	0.9	mgm.
Total sulphur	1.3 mgm. "	0.5	mgm.
Total phosphorus	1.9 mgm. "	0.9	mgm.

The analysis shows that we are dealing with an edematous fibrous tissue rather than with a myxoma, as shown by the presence of 90.53 per cent. of water and no appreciable mucin. The other figures merely confirm the fact that this tissue is more embryonic in character than the granuloma, as indicated by the greater amounts of sulphur, phosphorus and purin nitrogen.

The figure for fat was lower than anticipated, showing that the lipomatous elements did not form so large a proportion of the tumor as the gross appearance suggested. Nevertheless, there was present nearly two pounds of fatty material and four and a half pounds of protein in this tumor, despite the extremely emaciated condition of the host. Both microscopic and chemical examination afford striking evidence that a malignant tumor can retain considerable

amounts of fat under conditions in which the normal fat depots give up almost their last molecule of fat.

SUMMARY. There is reported the microscopic and chemical examination of a retroperitoneal liposarcoma without myxomatous elements, weighing sixty-nine pounds, being the largest solid tumor of which we can find record. It illustrates the capacity of malignant tumors to store up protein and fat, despite extreme emaciation of the host.

VASCULAR REACTIONS IN VASCULAR HYPERTENSION.[1]

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(From the Medical Clinic of the Peter Bent Brigham Hospital.)

A STARTLING observation, made two years ago, was directly responsible for the following study:

The patient was a woman, aged forty-eight years, with an extremely high blood-pressure and a normal renal function, one of those cases familiarly known as "vascular hypertension." Her systolic pressure had just been found to be 270, when the patient complained of rather severe cardiac pain. Nitroglycerin, gr. $\frac{1}{100}$, was given for relief. Curiosity led me to watch its effect on the blood-pressure. I was very much disturbed to find the pressure falling rapidly until it reached 110. In the meantine the patient had become very uncomfortable and was almost in collapse. An intravenous injection of adrenalin restored the pressure as quickly as it had fallen, and the patient become more comfortable.

This observation was so amazing that I was stimulated to study the reaction to nitroglycerin of the bloodvessels in this type of case. (I should state, at this point, that the above observation was unique on my part, none of its kind having since been made.) In the course of the investigation on the effect of nitroglycerin it was noted that during the preliminary period of our experiments, while we were trying to establish a base line before giving the drug, there was often a marked fall in pressure, due to rest and quiet. If exciting noises occurred near the patient, however, the pressure often rose to a surprising degree. The variations in pressure were so marked that we were led to determine not only the effect of nitroglycerin but also that of rest, excitement, exercise and afterward adrenalin. These results were interesting and instructive enough to warrant this report.

While some of the earlier experiments were, for obvious reasons,

[1] Study made under a grant from the Proctor Fund of Harvard University.

not done under standard conditions, the majority of those reported were carried out in the following manner: The patient was placed in bed in a quiet room for twenty to forty-five minutes. The object of this was to put him at his ease. At the end of this preliminary period the systolic and diastolic pressures and the pulse-rate were noted at five-minute intervals. Usually these patients established their base lines in thirty to forty-five minutes. Then I endeavored to excite the patient by urging him to talk about some disturbing subject. Generally his "high blood-pressure" (on which most of these patients are terribly centered) or his irritability, or some similar subject, was a sufficient excitation. It would, perhaps, have been somewhat better to have some standard form of excitation, such as that used by Meakins and Wilson in their study on "Irritable Heart,"[2] but no such means seemed practical or necessary. During the short two- or three-minute period of excitation, readings were again made and then the patient was allowed to become quiet again. After a sufficient time had elapsed to re-establish the base lines the effect of exercise—usually lifting from a reclining position to a sitting one ten times—or the effect of nitroglycerin under the tongue was tried. The adrenalin test was carried out in a similar way on another day. The doses of the drugs are given in the tables. Each observation consumed from one and a half to three or more hours on two days.

The first reaction to consider—the effect of quiet and rest—can be followed in Table I.

Here it can be seen that in the large proportion of the cases there was a definite and marked fall in both systolic and diastolic pressures during the preëxcitation period. In a very few, however, there was essentially no fall. In the systolic pressure the variations in the curves ranged from 0 to 46 mm., with an average of 21 mm. In the diastolic the fall varied from 0 to 18 mm., with an average of 10 mm. The time necessary to establish such falls varied markedly, as did the degree of fall, both being largely dependent on the nervous tension of the patient. The limits were from five to seventy minutes.

I presume that the following sequence of events took place in these patients. In spite of the preliminary rest period many were very nervous when the blood-pressure was first determined. As a result the pressure rose above its previous undetermined level. As the patient became accustomed to this maneuver the pressure fell. That this was true is indicated by the fact that the second and third readings were occasionally higher than the first. These were followed by the usual fall.

The effect of excitement showed a greater change in the pressure curves in the opposite direction. In every case there was a sharp rise. This was generally greater than the corresponding fall in the

[2] Heart, 1918, No. 1, vii, 17. In this study a pistol shot was used as a means of excitation.

preliminary period. The variations in the systolic pressures ranged from 10 to 52 mm., with an average of 30 mm. The diastolic pressures showed similar but less marked rises varying from 8 to 24 mm., with an average of 12 mm. In general, the rise was a very sharp one, the height of pressure being reached at the first determination. In some there was a rise at this reading, but the curve continued to ascend, reaching its maximum in ten to fifteen minutes.

TABLE I.—EFFECT OF REST AND EXCITEMENT IN VASCULAR HYPERTENSION.

Med. No.	Blood-pressure at start.		Maximum fall in mm. due to rest.			Maximum rise in mm. due to excitement.			Stimulus.
	Syst.	Diast.	Syst.	Diast.	Time in min.	Syst.	Diast.	Time in min.	
8022	126	98	3	18	40	10	8	5	Talking about a quack.
53819	148	92	30	16	65	24	8	15	Talking about father's death, etc.
7761	150	84	10	4	70	24	10	10	Talking about self and school work.
11704	152	98	14	2	20	34	24	15	Talking about blood-pressure and operation.
10664	172	98	18	2	20	40	10	5	Talking about her family.
6596	186	98	−1	5	30	20	8	5	Talking about her invalid daughter.
8976	186	110	0	0	—	52	10	5	Talking about her irritability.
8920	192	98	12	0	5	28	9	10	Thinking of a dead pet cat.
9799	198	99	23	10	40	—	—	—	Rest only.
9799	202	80	46	18	50	28	16	5	Talking about her school.
7608	209	92	35	10	60	50	10	5	Talking about her divorce.
8742	214	128	14	8	25	19	10	5	Talking about blood-pressure.
9846	222	122	—	—	—	36	8	10	Talking about self.
9846	227	122	19	12	20	32	24	15	Talking about blood-pressure.
6877	230	108	27	14	30	28	12	5	Talking about an attack of angina.
11581	238	104	26	1	65	16	9	10	Talking about blood-pressure.
Extremes	..	..	0–46	0–18	5–70	10–52	8–24	5–15	
Average	..	..	21	10	..	30	12		
					Controls.				
Manuel	114	64	4	4	10	14	10	5	Talking about exam.
F. Smith	116	62	6	2	20	14	6	5	Talking about exam.

In this and subsequent tables some of the cases do not seem to be hypertensive. They are, however, patients who have been many times hypertensive. By "time in min." is meant the time elapsed in reaching the maximum rise. The averages at the bottom of the table are only of those showing a positive rise. Two normal cases are included at the bottom of the table for comparison.

The effect of exercise in these hypertensive cases (Table II) showed somewhat conflicting results, as in normal cases. In general there was a fairly definite rise in both systolic and diastolic pressures. In one there was no rise in either. In two more there was a fall in the systolic and in five there was a fall in the diastolic pressure. The usual type of curve was a sharp rise followed by a fall almost as sharp. Rarely this is reversed. The results are similar to what occurs in normal individuals. The psychic element undoubtedly

plays a part in the reaction. The results are of special interest, however, in that they show how marked and how sudden a change may take place in these hypertensive patients as a result of moderate effort. A rise of 57 mm. in a normal patient is of much less significance than in a patient who already has a high pressure. (See Case No. 6596.)

TABLE II.—EFFECT OF EXERCISE.

Med. No.	Blood-pressure before exercise.		Maximum rise from exercise.		
	Systolic.	Diastolic.	Systolic.	Diastolic.	
53819 . . .	120	70	0	0	
8533 . . .	128	—	−4	—	
10664 . . .	132	84	10	−6	
7761 . . .	140	92	20	−2	
11704 . . .	142	100	16	0	
9799 . . .	162	80	26	8	
7608 . . .	168	82	45	0	Somewhat tired.
9799 . . .	170	80	42	4	
6596 . . .	184	98	57	22	
8976 . . .	186	120	10	−4	Tired.
10032 . . .	194	112	32	18	
8742 . . .	202	118	8	4	
6877 . . .	218	104	30	18	Tired.
8846 . . .	222	122	−20	−10	On repetition later; no fall in pressure from exercise.
Extremes . .	..	..	−20-70	−10-22	

It is of interest to note that while the diastolic pressures in the above experiments were more stable than the systolic they were far from being fixed. A variation of 24 mm. in the diastolic pressure within a two-hour period is indeed surprising. (See Case No. 8846, Table I.)

The observations recorded above may be merely exaggerations of what occurs normally. But in the hypertensive case they are of vastly greater significance. Furthermore the recognition of the fact that such wide variations in pressure can occur within a comparatively short period due to such commonplace causes as those used, is of extreme importance in estimating the meaning of any given high pressure. It must, too, make us very cautious about the interpretation of the effects of treatment. If a patient's pressure under the influence of mental and physical rest can drop 35 mm., and then under the excitation of talking about family difficulties can rise again 50 mm. (see Case No. 7608, Table I; see also same case, Table II) one can readily understand the wide variations that must take place every day in this type of case. One can understand, too, of how little significance are even moderate changes noted from time to time unless they are all in the same direction.

As a result of such observations as these I have become very skeptical about the therapeutic value of drugs, electricity, baths, etc., in this type of case. If they produce any effect at all it is largely through the accompanying mental or physical rest imposed.

Unless one can show a permanent or at least a long-continued lowering of pressure of a considerable degree, one cannot rightfully claim that any given therapeutic maneuver has been beneficial. I have yet to see a fall in pressure resulting from any method of treatment (except rest) that cannot be duplicated or exceeded by those observations we have made on rest. By the latter means one may reduce the pressure markedly, but, unfortunately, on the resumption of effort it rises again. However, with care it need not rise to its former level. After all, this is as much as we should reasonably expect, except in the earliest cases.

Another thought arises out of this study. The extreme liability of the vasomotor system in this type of case, as indicated by the marked and sudden changes in pressure, makes us wonder what the effect on the vessels must be. Will not these patients show earlier and more marked sclerosis of the vessel walls?

TABLE III.—EFFECT OF NITROGLYCERIN.

Med. No.	Blood-pressure at start.		Primary rise in mm.		Primary fall in mm.		Time in min.	Secondary fall in mm.		Time in min.	Dose in G.	
	Systolic.	Diastolic.	Systolic.	Diastolic.	Systolic.	Diastolic.		Systolic.	Diastolic.			
53819	148	92	4	0	—	—	—	2	16	15	0.0006	Slight reaction.
11704	152	98	22	22	—	—	—	2	0	30	0.0009	
10664	154	100	20	2	—	—	—	22	18	35	0.0009	
6596	186	98	20	4	—	—	—	5	6	35	0.0009	
8976	186	110	14	16	—	—	—	18	8	30	0.0009	
9799	188	99	24	11	—	—	—	19	14	50	0.0009	
9767	190	112	19	10	—	—	—	3	2	15	0.0006	
7608	209	92	26	10	—	—	—	22	8	45	0.0009	
6877	230	108	11	21	—	—	—	6	0	20	0.0009	
9106	238	154	5	16	—	—	—	25	0	20	0.0009	
10962	288	172	10	—	—	20	15	20	—	10	0.0009	
7761	128	92	—	—	16	42	23	—	—	—	0.0009	Some doubt about diast. pressures.
9767	190	112	—	—	2	2	5	−8	−10	15	0.0006	See above 9767.
8920	196	120	—	14	28	—	14	60	31	244	0.0006	
8880	212	102	—	14	32	—	23	—	4	—	0.0006	
11581	238	104	—	12	22	—	7	—	—	—	0.0009	

This table is divided into two parts. The first 11 cases are those which showed a primary rise. The others showed a primary fall. The last three in the second group showed no primary rise in the systolic pressure but a rise in the diastolic. One case in the first group showed a rise in systolic pressure and a fall in the diastolic.

In studying the effect of nitroglycerin in this disease we received two genuine surprises (Table III). Instead of finding a sharp fall of pressure after a fairly large dose of this drug placed under the tongue we found that the majority of our cases showed a primary rise. This, too, occurred during the height of the symptomatic reactions to the drug. The patient was flushed. There was pounding in the head and in the region of the heart. The radial pulses were definitely more full, and yet the systolic and diastolic pressures

were higher at this period instead of lower. Furthermore, these rises were not always trifling, being over 20 mm. in several cases. I am of the opinion that in these strikingly nervous people the excitement produced by the nitroglycerin caused the primary rise.

The second surprise was in the comparatively slight depressor effect of the drug. The drop in pressure, either following the primary rise or when there was no such rise, was comparatively slight. I say this in spite of the apparent contradiction in Table III. If one notices those cases in which a considerable fall took place after the nitroglycerin, he will see that, for the most part, it required a very long time to reach this depth. I cannot help feeling that much of this fall must be attributed to rest and quiet, otherwise, it seems to me, we should obtain the greatest drop during the period of vasodilatation, *i. e.*, during the period of flushing, increased pulse volume, etc.

Observations like these confirm our ideas about the uselessness of nitroglycerin in hypertension. Formerly we discarded this drug on the grounds that the depressor effect was too transient to be of value. These experiments of ours show that there is but little reduction of pressure due to the drug and that there is often a primary rise in pressure. Rest alone is far more effective.

TABLE IV.—EFFECT OF ADRENALIN.

Med. No.	Blood-pressure at start.		Maximum rise in mm.		Time in min.	Duration of rise.	Dose in c.c.	Remarks.
	Syst.	Diast.	Syst.	Diast.				
7761	132	90	42	50	2	20	0.5	
8533	134	48	48	10	10	45	0.5	Plateau curve.
53819	140	80	30	12	2	60	0.5	Rather prolonged rise.
11704	160	104	80	−40	2	25	0.5	Sharp, marked rise.
10664	166	98	52	10	2	22	0.5	Double rise.
7608	194	90	40	10	24	120	0.5	Slow rise and fall; typical syndrome reaction.
6877	199	94	70	22	5	40	0.5	Double rise; angina.
8976	200	120	26	10	10	15	0.25	Too small a dose.
6596	206	104	87	22	2	95	0.5	Sharp rise and fall, the latter trailing off; angina.
8846	214	118	8	−2	2	6	0.5	? about site of injection.
Extremes	..	..	26–87	10–50	..	15–120		
Average	..	..	53	18				
					Controls.			
Manuel	114	62	8	2	4	55	0.5	Normal student.
De Blois	100	60	14	10	2	50	0.5	Gastric necrosis.

Only 2 normal cases are included at the bottom of the table for purposes of comparison. For normal controls, see report of work at Lakewood.

In spite of the above, nitroglycerin has a place in the treatment of one of the complications of vascular hypertension. Clinical experience has taught us its value in angina pectoris. In the light of our results one cannot help wondering about the effect of this drug on the various bloodvessels of the body. There is no doubt that it

causes a vasodilatation of many of the vessels. This can be seen and felt. No doubt, too, in angina the relief comes from such a vasodilatation, and yet there is a compensating or overcompensating mechanism in this disease which maintains the pressure at a high level in spite of the nitroglycerin.

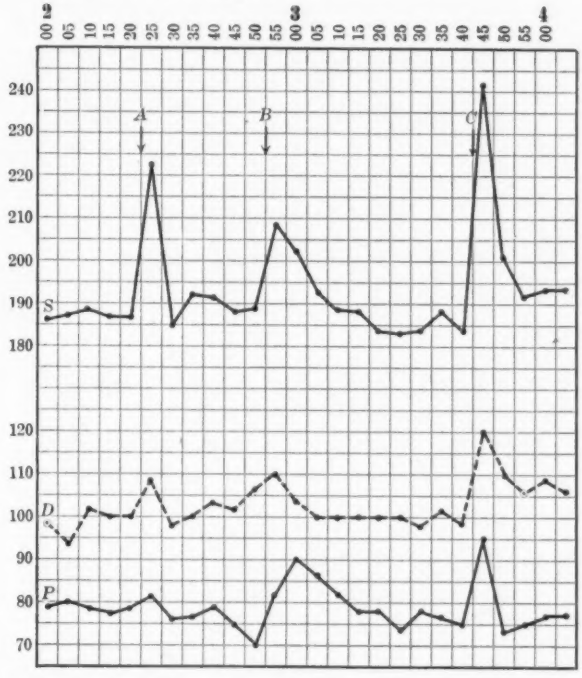

CHART I.—In this chart and those that follow, the ordinate indicates time, the abscissa, pressures, pulse and respiration rates. *S*, systolic blood-pressure; *D*, diastolic blood-pressure; *P*, pulse-rate; *R*, respiration-rate. In the diastolic pressure curve the symbol ○ is occasionally used. This indicates that no abrupt change from a sharp to a muffled note was heard but merely a gradual decrease in sounds. The ○ represents the level at which the last sound was heard. Chart I demonstrates the effect of emotion, nitroglycerin and exercise. At *A* is noted the effect of talking two minutes about an invalid daughter. At *B* nitroglycerin, 0.0009 G., was given under the tongue. Note especially the rise of 20 mm. in the systolic pressure and the comparatively negligible fall of this pressure. At *C* the patient lifted herself from the reclining to the sitting position ten times.

One of the most interesting phases of this study of ours is that of the effect of adrenalin, as in the Goetsch test.[3] Sturgis and Wearn,[4] at Lakewood, have shown that ½ c.c. of a 1 to 1000 solution of drug given intramuscularly in normal individuals causes no appreciable rise in blood-pressure.

[3] New York State, Jour. Med., July, 1918, xix, 259.
[4] Arch. Int. Med. September, 1919, xxiv, 269.

Our data is very meager consisting of only ten cases. This small number is due to the fact that we became afraid to use the drug in this type of case. The reaction was so striking and so nearly constant that we feel it worth while to report our cases. In only one case (No. 8846, Table IV) did we fail to get the typical reaction. In this one case there was some doubt about the technic.

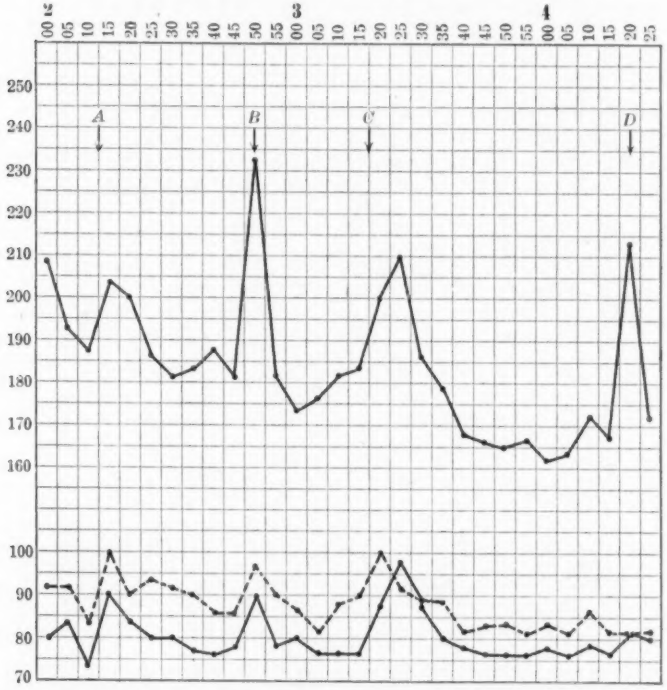

CHART II.—This demonstrates the effect of emotion, nitroglycerin, and exercise. At *A* the patient was stirred up while her pressure was apparently falling, by placing a blanket on her chest. At *B* can be seen the effect of talking two minutes about her divorce. At *C* nitroglycerin, 9.0009 G., was given under the tongue. At *D* can be seen the effect of lifting herself from the reclining to the sitting position ten times.

The technic was almost identical with that used by Sturgis and Wearn[5] at Lakewood, the only difference being that we used the prepared solution of adrenalin, whereas there a solution was made from tablets. The patient was kept at rest in bed in a quiet room for one-half to one hour. Control pressure readings, pulse and respiration rates, symptoms, etc., were then noted at five-minute intervals until a base line was established. Then ½ c.c. of a 1 to 1000

[5] Sturgis and Wearn, cited above.

solution of adrenalin was injected into the deltoid or biceps muscle. Pressure readings, etc., were thereafter made every two minutes for ten minutes, then every five minutes for one hour or more, and then every ten minutes until the pressure came back to normal.

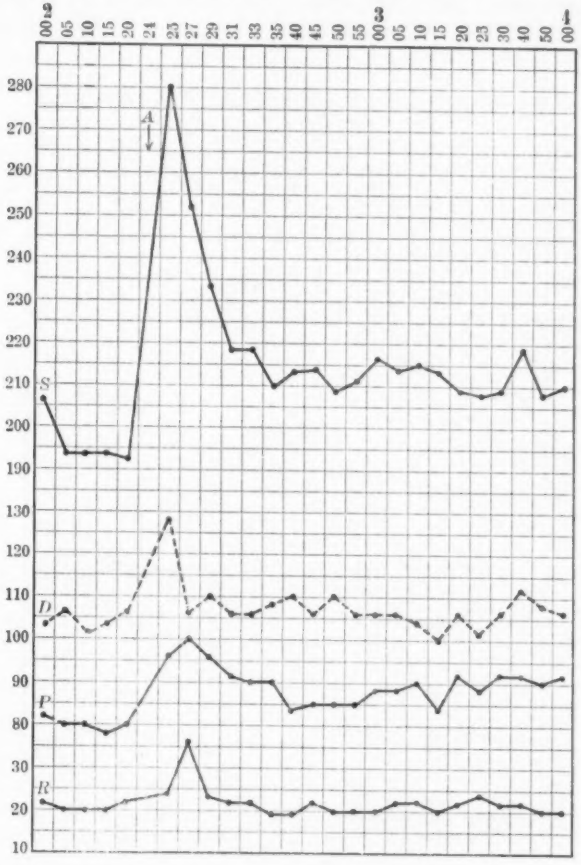

CHART III.—This demonstrates the effect of adrenalin. At *A* the patient was given 0.5 c.c. adrenalin (1 to 1000) intramuscularly. Note the sharp, terrific rise in pressure.

The reaction to the drug was striking and in some instances alarming. A typical one showed a sharp rise of pressure averaging 53 mm. for the systolic and 18 mm. for the diastolic. In two cases the maximum systolic rise was over 80 mm. When such a rise took place within two minutes after the drug was injected it was indeed striking, especially if it started from a base line of over 200 mm.

Generally the pressure was not maintained at the high level for longer than a few minutes. It then fell, sometimes fairly abruptly and sometimes more gradually. Occasionally there was a secondary slight rise during the fall. The time required to reach the maximum pressure and to reach the former level is indicated in Table IV. Immediately after the injection of the drug the patient became pale

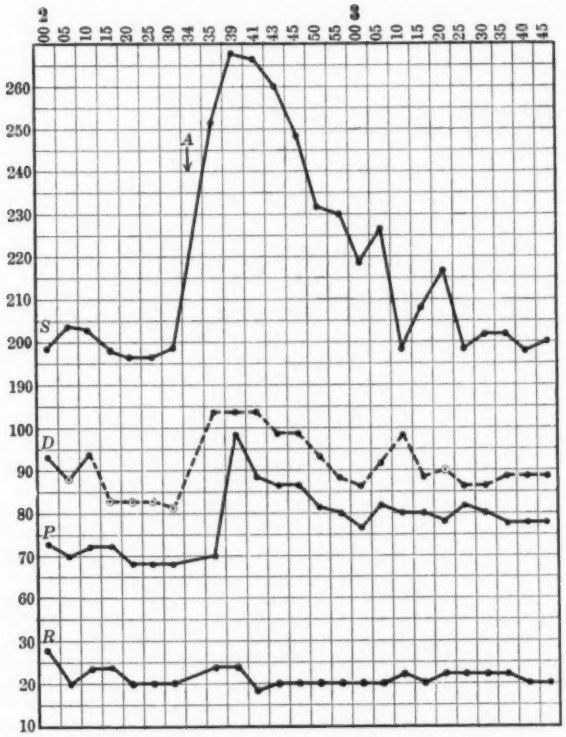

CHART IV.—Another adrenalin effect. At *A* the patient was given 0.5 c.c. adrenalin (1 to 1000) intramuscularly. Patient had severe angina pectoris.

and tense and had a feeling of tingling all over. There was sometimes a slight tremor of the hands and usually a pounding in the head and in the region of the precordia. In one or two there were practically no symptoms.

Neither in the symptomatology nor in the pressure curves did our cases resemble those of the "effort syndrome" reported from Lakewood. Only one case (No. 7608, Table IV) showed the typical "syndrome curve," but this patient did not display the symptoms which are so characteristic.

A few of the curves resemble more closely those reported by Goetsch in his hyperthyroid series, especially those with the secondary rise (Nos. 10664 and 6877, Table IV). As a rule, however, our cases showed a much more abrupt and higher rise than did those of Goetsch. That these patients were cases of hyperthyroidism is not at all credible. In fact, we have determined the basal metabolism on a few that seemed possibly thyrotoxic and found it to be subnormal instead of raised.

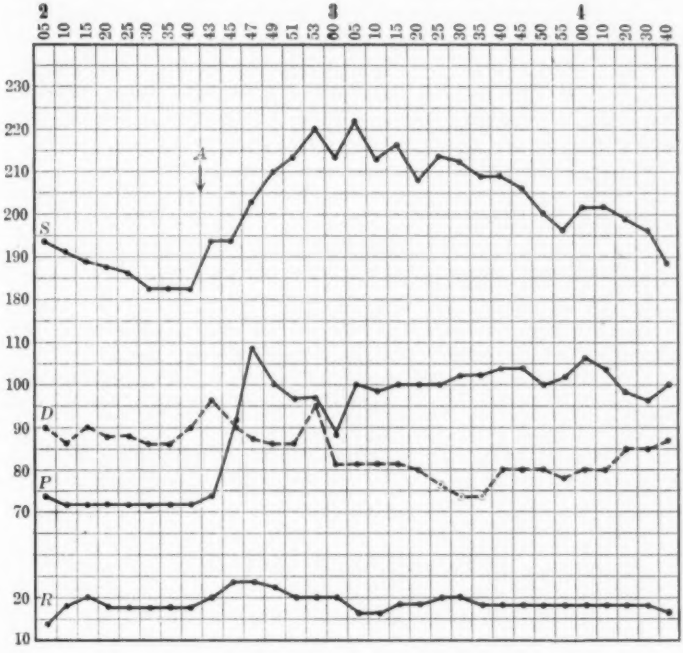

CHART V.—Another adrenalin effect. At *A* patient was given 0.5 c.c. adrenalin (1 to 1000) intramuscularly. Note the more slow and sustained rise in pressure. This resembles the reaction obtained by Goetsch in his hyperthyroid cases.

Two cases in this series warrant special comment because of the alarming nature of the reaction. Case No. 6877 was an elderly woman who had had several attacks of rather mild angina pectoris. After the injection of adrenalin the pressure rose immediately 70 mm. The patient became very pale and frightened and had the worst attack of angina she had ever experienced. Fortunately the pressure fell rather promptly and the symptoms abated as quickly.

The second case (No. 6596) showed a similar alarming jump in the systolic pressure from 193 to 280 in one minute. She, too, became very pale and frightened and had a tremendous sense of constriction in the region of her heart.

Both of these patients felt they were going to die and I was much alarmed. It was a great relief to see the pressure fall instead of continuing to rise.

Cases like these made me unwilling to try this reaction further in patients with a pressure over 190 mm. Consequently my series is small. They should serve as a warning against indiscriminate use of adrenalin intramuscularly.

The "adrenalin reactions" in hyperthyroidism, the effort syndrome and in vascular hypertension are interesting problems requiring much more study. In the last group the worker must realize that he is "playing with dynamite."

It is safe to conclude from these studies that:

1. The vasomotor system in vascular hypertension is extremely labile and sensitive.
2. Mental and physical rest causes a marked fall in pressure.
3. Excitation causes a more marked abrupt rise.
4. Exercise usually causes a similar rise.
5. Nitroglycerin produces practically no fall in pressure and there is often a primary rise following its absorption.
6. The vessels are especially sensitive to the intramuscular injection of adrenalin, a marked rise in pressure taking place immediately after its injection.

I gratefully acknowledge the assistance of Miss Elsa Griffin.

THE DANGERS OF ASCARIASIS.

By Bowman Corning Crowell, M.D.,
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Animal parasites of the human host are today probably attracting more attention than ever before, while with improved modern methods new ones are being discovered, and, as the result of accumulated knowledge, pathogenic properties are being ascribed to those formerly considered harmless. The technic of the laboratory is being employed in all its refinements to aid in diagnosis, and, on the other hand, treatment of some forms of intestinal parasitism is being carried out on a more scientific basis and on a larger scale than could have been foreseen only a few years since. International campaigns against parasites are now taken in a matter-of-fact way, and the mention of their scope in all their vast inclusiveness excites little more than passing admiration.

Probably greater efforts have been expended against the hookworm, the malarial parasite and the entameba than against the other animal parasites. But who ever gives a thought, other than one of disgust, to the *ascaris?* And yet the wide territory covered by the *ascaris*, the number of the population infested by it and the harmful results attending its presence entitle it to more respectful attention than is usually accorded it. In practically all places, if not in all, in which examinations are made for parasitic infections it is found that the *ascaris* is the parasite most frequently encountered. It is also one of the worms that is most easy of recognition, and for that reason very little difficulty attends the diagnosis. Perhaps these facts have helped to distract the interest of the physician from this homely worm. The average physician who has had any experience in tropical countries where the *ascaris* abounds will say that the treatment of ascariasis is easy, which is probably true in the vast majority of cases, and that its harmful effects are slight anyway, which is decidedly not true. It is my opinion that the word of the pathologist on this subject is heard only too infrequently, and it is for this reason that I wish to put on record some of my experiences with this worm as seen in the autopsy room during several years in Manila. This experience has led me to look upon the *ascaris* with more than the usual amount of respect and fear. The pathologist and the medical man who follows his cases to their ultimate diagnosis are better qualified to speak on this subject than the pure parasitologist or the man who knows only mortality statistics based upon clinical diagnosis alone. A simple examination of the hospital records of tropical countries will show what a large number of cases are admitted to these hospitals for ascariasis alone, aside from those in which ascariasis is found complicating other diseases which are usually considered more serious. If this be followed by an examination of the records of the associated morgues, with their never-ending list of surprises, the case against the *ascaris* will be strengthened. I regret not being able to give accurate statistics on this subject, but I know that they would convince even the most skeptical that this parasite has a very decided economic importance as judged from the morbidity caused by it.

As the worm is a large one and easy of recognition, much attention was paid to it by the earlier workers on this subject, and some of the older books on parasitology give a better idea of the importance of this worm than one would acquire from a perusal of some of the more modern works. The average medical student at present is therefore sent out into practice with an erroneous idea concerning the importance of this disease. Fortunately, attention has recently been again directed to this worm through the work which is being done on the method of transmission of the parasite, and it therefore seems an opportune time to once again draw attention to the harm attending infestation by it.

Fantham, Stephens and Theobald,[1] in their recent book, *Animal Parasites of Man*, and Blanchard,[2] in his *Zoölogie medicale* of thirty years ago, give excellent ideas on the pathology of ascariasis, and frequent references will be made to these works.

PREVALENCE OF THE PARASITE GEOGRAPHICALLY AND IN MAN. Documentary evidence will not be given here to bear out the statement that the *ascaris* is more widely distributed from the geographical standpoint and infests a larger number of people than any other known intestinal parasite. Text-books, monographs, hospital and morbidity records all agree upon this point and place it beyond all possibility of dispute.

My personal experiences with ascariasis were chiefly gained in the Philippine Islands, and I shall quote a few statistics from there to show its incidence. Willets,[3] in 1911, found 62.3 per cent. of 4278 persons infested with *ascaris* as compared with 54.37 per cent. with hookworm, and in a grand total of 19,302 examinations compiled by the same author in the Philippines, 61.36 per cent. had *ascaris* and 30.57 per cent. had hookworm. In some of the series from which these statistics were compiled the percentage of *ascaris* infestations was as high as 83. In 500 consecutive autopsies on cases the majority of which came from a hospital in which routine treatment for intestinal parasites was practised, Crowell and Hammack[4] found 41.2 per cent. with *ascaris* as compared with 16.6 with hookworm. In other places the incidence of the *ascaris* runs up to almost 100 per cent.

The *ascaris*, unlike many other parasites, does not confine itself to the tropics, but is found in all climates and in all latitudes.

PATHOLOGICAL EFFECTS OF THE ASCARIS. The pathological effects of the *ascaris* are numerous, but are susceptible of classification.

I. Mechanical effects are prominent and certain from the *ascaris;* these may be exerted when the worm is in the intestine or in the course of its migrations.

II. As a carrier of infection the worm is also of importance in its migrations. Whether through its eggs it may prove a source of transmitting infection is not yet proved.

III. The larvæ cause a bronchopneumonia in experimental animals, and it is not improbable that they may do so in some cases in infants.

IV. The presence of a harmful toxic substance is a source of danger and reflex nervous symptoms due either to mechanical or toxic irritation are also frequently encountered. These phenomena will now be dealt with in more detail.

[1] Animal Parasites of Man, 1916, John Bale, Sons & Danielsson, Ltd., London.
[2] Traite de Zoölogie médicale, 1889, J. B. Baillière et Fils, Paris.
[3] Philadelphia Jour. Sc., Sec. B, 1911, vi, 77.
[4] Ibid., 1913, viii, 157.

I. Mechanical Effects of the Presence of the Ascaris. 1. *In the Intestine.* Large numbers of the worms may be present in the intestine; one individual has been reported to have evacuated as many as 600 worms in one day and other large numbers are frequently reported. Fauconneau-Dufresne is quoted by Blanchard as having published a case in which a boy, aged twelve years, evacuated more than 5000 worms in less than three years, the majority by vomiting; 600 were evacuated in one day only!

In our autopsy experience as many as 200 were found in the intestine of one child, and numbers from 50 to 75 were so frequent as to occasion no surprise. Such masses of these worms may cause not only constipation but absolute obstruction. Fantham, Stephens and Theobald credit cases of ileus to Mosler and Peiper, Raie, Rehberg, Rocheblave, Leichtenstern, Huber and Wilms.

The collection of large coils of the worms in the sigmoid flexure of the colon may cause palpable, firm masses in the left abdominal quadrant, which have led to the clinical diagnosis of true tumor of the intestine. This is a frequent source of error to newly arrived physicians in the tropics, and I have seen an abdomen opened under this false diagnosis. Vermifuges have caused many a prospective operation for suspected abdominal tumors to be indefinitely postponed.

The question as to whether the *ascaris* actually may perforate the intact wall of the intestine or stomach is still open. Certainly the worm is often found in the peritoneal cavity at autopsy. Some authors quoted by Blanchard attributed this wholly to the parasites, whether they produce the perforations or determine foci of gangrene. Others maintain that the perforations occur independently of the worms, and that they simply use an exit which they find available; and still others maintain that the worm is capable of passing through the intestinal membrane by simply separating the fibers; its passage completed these fibers come together again, owing to their elasticity and contractility, and all trace of the perforation disappears. Blanchard collected a total of 81 cases in which the *ascaris* had passed out through the body wall, especially at the umbilicus and groin. Davaine points out that these exits of the *ascaris* are more frequent at the umbilicus in children and at the groin in adults, corresponding to the frequency of hernia at the two places. They have also been reported as having been passed through the urethra with the urine, and here it may be supposed there was fistulous connection between the bladder and the intestine.

Other wanderings of the worm have been described in which it would seem that the worm had passed through tissues. It seems probable that exact anatomical investigations would increase the number of cases in which it would be found that these cases are explicable on the basis of fistulous openings in the tissues through which the worms may pass. These fistulas may remain patent or

close after the passage of the worms. The predilection that these worms have for passing into small orifices is well known and frequently emphasized. Thus, Clason[5] records that in the case of an idiot whose custom it was to swallow glass beads the *ascarides* showed a predilection for sticking in the beads and were passed in the feces. Blanchard refers to the fact of the worms engaging themselves in the holes of buttons that have been inadvertently swallowed, and says that certain authors have even proposed objects of this kind as worm snares. This habit of the worms was exemplified on one of our cases in which a full-grown *ascaris* had penetrated through the tunnel in the submucosa, connecting two adjacent tuberculous ulcers of the colon, and was found protruding from both ulcers and doubled on itself so as to form a knot.

I have seen the *ascaris* in the peritoneum at autopsy many times after cases of rupture of the intestine, and in some cases when there was a postmortem digestion of the stomach, which made an easy exit possible. More interesting are the cases of perforative appendicitis or diverticulitis through which the worm has passed to the peritoneum. Several cases of perforation of the appendix occurred in our autopsy service in which *ascarides* were present in the peritoneum. It is natural to suppose that they had passed out through the opening in the appendix, but this does not indicate that they were the cause of the appendicitis. I have notes of four appendices removed surgically which contained the *ascaris*, and in none of these was there evidence of severe disease in the appendix. These cases are as follows:

A Filipino woman, aged thirty-two years, had the appendix removed incidental to the removal of a large multilocular cyst of the ovary. The appendix contained an *ascaris* but showed no pathological change. A similar case was seen by me in New York ten years ago.

A Filipino woman, aged twenty-seven years, had the appendix removed incidentally to a cholecystectomy; it contained an *ascaris*, and slight atrophy of the mucosa was present.

A Filipino woman, aged forty years, had the appendix removed under the diagnosis of chronic appendicitis; its lumen contained a portion of an *ascaris* and two trichurides; slight catarrhal changes only were present.

An American child, aged seven years, had the appendix removed under the diagnosis of acute appendicitis. The appendix measured 7.5 cm. in length and about 7 mm. in diameter; the superficial bloodvessels were markedly congested and the lumen contained an *ascaris*. Microscopically there was seen only a slight atrophy of the mucosa.

In the latter 2 cases, and in other similar ones that I have seen, it is probable that the appendicular symptoms would have been

[5] Quoted by Fantham, Stephens and Theobald.

transitory. The reason for my mentioning these cases is to emphasize that the presence of the *ascaris* in the appendix does not necessarily lead to severe changes in the appendix, and also that symptoms simulating appendicitis may be caused by their presence. It is, however, probable that the pain is the most prominent symptom and that a temperature record with a blood count and examination of the feces might clear up the diagnosis. Another case illustrating another point in this connection will be reported.

A male Filipino of four and a half years was autopsied seven and a half hours after death. The abdominal cavity contained about 50 c.c. of thick, greenish, creamy pus and the coils of intestine were matted to one another by delicate fibrinous adhesions. The coils of the small intestine were much dilated and the serosa was pale except in the areas covered by the fibrin. Lying free on a coil of the small intestine next to the anterior abdominal wall to the left of the umbilicus was a small *ascaris*. Other *ascarides* were found enmeshed in the adhesions, especially in the region behind the transverse colon and over the region of the left kidney. There were in all eight living *ascarides* found free in the peritoneal cavity. Over the region of the left kidney one coil of intestine presented a diverticulum which had perforated. This was situated in the ileum at a point 30 cm. from the ileocecal valve, and originated from the lateral aspect of the intestinal wall nearer to the mesenteric attachment than to the opposite side. At its tip were two small perforations covered with fibrin. It is not necessary to suppose that the perforations were caused by the *ascaris*, but it is very easy to imagine that they might have determined a perforation in an inflamed diverticulum, and it is certainly true that their passage from the diverticulum prevented the circumscribing of the inflammatory phenomena by the usual process.

I have had several cases in which the *ascaris* had actively opened up a repaired wound of the intestine, thus determining a fatal peritonitis. This emphasizes the importance of the routine examination of the feces and the use of vermifuges if *ascaris* is found in all cases of abdominal operations of choice. It is unfortunate that this is a precaution often omitted even when *ascaris* is known to abound. Of course there are cases in which vermifuges are contraindicated and cases in which operation is urgent. In these cases precautions appropriate to the individual case should be taken. I will here report one case that I have had among others illustrating this danger.

No. 5695. A male Filipino, aged thirty-seven years, received a severe blow on the abdomen from a "push-cart." He was taken at once to hospital, where intestinal rupture was found and repaired by end-to-end anastomosis. Death ensued eight days after operation and autopsy was performed six hours after death. The following extract is made from the autopsy protocol:

On the abdomen, at the margin of the right rectus muscle, between the umbilicus and pubis, is a long surgical incision enclosed by sutures. From the lower end of this there protrudes a gauze and rubber drain. On removing the sutures the margins of the skin are easily pulled apart, and on separating these margins the subcutaneous tissues are found to be necrotic and bathed in foul-smelling pus. The sutures below the skin have separated and a loop of intestine presents in the abdominal wound, forming its base. This loop of intestine is intimately adherent to the anterior abdominal wall by fibrin. On opening into the abdominal cavity the entire left side of the abdomen is found free from exudate. On the right side the loops of small intestine are adherent to the anterior abdominal wall and to one another. The intestine in the upper part of the abdomen is markedly dilated. A loop of small intestine is present in the middle of the right side of the abdomen, with its surfaces closely adherent to one another. On separating these adhesions an opening in the intestine is found which had been closed by sutures. From this opening fecal matter pours and two dead *ascarides* lie on the mesentery adjacent to this opening. The great omentum is pulled downward and to the right, and is adherent by fibrin over the matted intestine. On loosening the loops of the intestine and cecum from the right abdominal wall there is found a large collection of pus which fills the lower right quadrant of the abdomen and extends into the pelvis. Between the middle portion of the ascending colon and the right abdominal wall there is found a dead *ascaris* lying on the mesentery just behind where the duodenum pierces the mesentery. In both of these places fibrin is piled up from the peritoneum about the *ascarides*, almost enclosing them, so that when the worms are removed the groove of fibrin on the peritoneum remains. On separating all the adhesions of the intestine and mesentery and removing them it is found that the intestine is dilated down to within about half a meter of the cecum, and below this it is collapsed. The opening in the intestine is at a point 2½ meters from the pylorus. Here an end-to-end anastomosis has been performed, but the margins were separated. In the upper intestine brown, watery fluid is present and about a dozen *ascarides*. In the collapsed lower intestine there is abundant, dry, sticky feces and some trichurides are found in the cecum.

Interest attaches to the presence of the *ascarides* in the peritoneum and the reaction of the peritoneum to their presence as has been described above. At the time of operation fecal matter was present in the abdomen and the peritoneum was thoroughly flushed with salt solution. It is a question whether the *ascarides* entered the abdomen before or after operation. The absolute localization of the extensive peritonitis to the right side of the abdomen is also of interest, as is the point of rupture of the intestine. It is extremely probable that the peritoneum was thoroughly cleaned at the opera-

tion and that subsequently the *ascarides* had forced their way between the sutures, thus causing the fatal peritonitis. The reaction of the peritoneum about the worms proves that this was not a case of postmortem migration. Microscopically ova of *ascaris* were abundant in the peritoneal exudate.

2. *Outside of the Intestine.* When, at autopsy, the *ascaris* is found outside of the intestine there is frequently some difficulty in determining whether the migration occurred before or after death. Unless there is some clinical evidence before death of the *ascaris* being outside of the intestine I have made it a rule to consider the migration to have occurred after death, except in those cases in which there is evidence of reaction to the presence of the worm or when the worm is in such a condition or position that it obviously could not have attained after death. For instance, in the case just reported there was no doubt that the migration was antemortem on account of the piling up of the fibrin about the individual worms. The presence of the *ascaris* in the larynx or trachea after death has not been accepted by me as evidence of the cause of death unless other evidences such as those caused by asphyxia were also present. At times dead worms are found in the bile ducts or in other positions when the short period elapsing between the death of the patient and the autopsy has rendered obvious the antemortem occurrence of the migration. Such considerations are of great value in leading to correct interpretations of the autopsy findings.

Migration to the Bile Channels and Liver. Migration of the *ascaris* into the common bile duct and thence into the gall-bladder or into the intrahepatic bile ducts is a frequent occurrence, and must be much more frequent than is indicated by the reported cases, as the diagnosis is made only at operation or at autopsy. One of several worms may pass through the papilla of Vater in this way, and may cause occlusion, followed by icterus, with the symptoms of colic, or may lead to the development of a cholangitis and later abscesses of the liver. Thus the action of the worms in the bile ducts may be either purely mechanical or they may serve as carriers of infection into the liver. Leer[6] says that *ascarides* are the second most frequent cause of liver abscess. Vierordt[7] states that mature females can penetrate into the liver and there deposit eggs, and that such eggs appear occasionally to undergo segmentation. The worms may be found in the ducts alive or dead, and may show constrictions about the body when they have become strangulated by pressure.

After the death of the worms in the bile ducts they become macerated and flattened, stained with bile, and may become encrusted with bile pigments, as is shown in cases 3422, 2387, 2524, 3192, 3905. (See below.)

[6] British Med. Jour., 1916.
[7] Volkmann's Samml. klin. Vortr., No. 375.

The abscesses in the liver are usually multiple and the pus within them very foul. They may perforate in any direction and the worms may be evacuated through the opening formed. Communication of the abscesses with the bile ducts is usually readily recognizable on account of the dilatation of the ducts caused by the passage of the worms. If the abscesses be seated near the periphery of the liver adhesions may form between the liver and the adjacent tissues. In Case 1007 a subdiaphragmatic abscess had formed and the base of the right lung was adherent to the diaphragm, with a localized empyema. Lebert is quoted by Blanchard as having seen a liver abscess extend through the diaphragm and lung and evacuate itself through the bronchi, as sometimes occurs in cases of hepatic amebiasis. It is to be remembered that *ascarides* may migrate into the ducts of a liver which is the seat of abscesses from other causes.

In one of our cases to be reported (4424) there were three large *ascarides* in the hepatic ducts, the largest of which extended to the surface of the right lobe of the liver. The common duct was distended and practically occluded by the folded bodies of two full-grown *ascarides*, and one protruded from the papilla of Vater. There was thrombosis of the splenic vein along the pancreas and an acute hemorrhagic pancreatitis, with fat necrosis, without the presence of a worm in the pancreatic duct. It can only be a matter of conjecture in this case whether the pressure of the hilus of the liver from the worms in the ducts led to the venous thrombosis to which the pancreatitis was secondary or whether the pancreatitis resulted from the occlusion of the opening of the pancreatic duct into the common duct, and the thrombosis was a sequel to this.

Reports of some of our cases of migration into the bile passages follow:

CASES OF ASCARIS IN BILE DUCTS AND LIVER.

5684. *Ascarides* in intestine, stomach, esophagus, bile ducts and liver; probable asphyxia from *ascaris;* icterus.

A little flaxen-haired American-Filipino "mestizo" of four years was known to have *ascarides* and had undergone home treatment. One morning about a week after treatment had ceased he was taken *suddenly* with jaundice and sweating, both of which became intense. Within five hours he was in hospital and not apparently in a very serious condition; he was put into bed and the father left him to find the physician. Upon the father's return with the physician within five minutes the child was found dead. Autopsy was performed within two hours of death. There was no evidence of status lymphaticus. The skin had a decided icteric color. The blood was dark and not coagulated; the lungs were distended and there was a frothy fluid in the bronchi, trachea and larynx. The larynx was edematous. There were small epicardial and pleural hemorrhages. The duodenum was distended with numerous *ascarides*, which

formed a compact mass throughout its entire length, and there were numerous *ascarides* in the jejunum. The bile ducts were distended with five large *ascarides*, which extended into both hepatic ducts, obstructing the flow of bile. These living *ascarides* extended nearly to the surface of the liver. There were numerous *ascarides* in the stomach and six large ones in the esophagus. The only lesion found in the other viscera was congestion.

FIG. 1.—Ascaris tunnelling between two tuberculous ulcers of colon.

There appear to be two possible explanations of the sudden death in this case: (1) Reflex nervous phenomena from intestinal and biliary ascariasis, or (2) what seems more probable, asphyxia from *ascarides* in the upper respiratory tract. It is true that there were no *ascarides* found in the respiratory tract at autopsy, but the esophagus was filled with them, the larynx was edematous and there were other signs of asphyxia as noted above.

4424. *Ascarides* in stomach, hepatic and common bile ducts; hemorrhagic pancreatitis; thrombosis of splenic vein; fat necrosis.

Female, Filipino, aged thirty years. Duration of illness, two days. No jaundice; 100 c.c. of blood-tinged fluid in peritoneum. Very abundant extravasation of blood, behind the peritoneum, in pancreas and lesser peritoneal sac. Liver weighs 1255 grams and is of normal size. The hepatic duct contains three large *ascarides*, the largest of which extends to the surface of the right lobe of the liver. The common bile duct is distended and practically occluded by the folded bodies of two full-grown *ascarides*, and one protrudes from the papilla of Vater into the duodenum. The gall-bladder is considerably distended and contains brick-red, thin bile.

The pancreas lies in its normal position and is surrounded by a very hemorrhagic serous membrane. The pancreatic duct is free

and opens into the common bile duct, about 5 mm. above the papilla. The accessory duct is not seen. The splenic vein running along the pancreas contains a long thrombus, which is adherent to the posterior wall of the vein and seems to extend into the branches of the splenic vein leading into the substance of the pancreas. The body and tail of the pancreas are dark red, considerably softened and somewhat friable. The cut surface shows bright purple to dark red soft lobules which are separated by dark red septa. In some areas the lobules are yellowish, very soft, with intensely hemorrhagic zones surrounding them. The head of the pancreas is firm, pale and its cut surface shows light, yellowish, clearly outlined, firm lobules. On the surface of the capsule are seen a few grayish-white to yellowish-white, flat, pin-head-sized areas, which are also seen on the surface of the mesentery and omentum. Three large *ascarides* in the stomach.

FIG. 2.—Living and dead ascarides in intrahepatic bile ducts, liver and gall-bladder.

3422. *Ascarides* in bile ducts, stomach and intestine; multiple abscesses of the liver.

Female, Filipino, aged five years. Duration of illness, one month. Autopsy performed nine and a half hours after death. Death from pulmonary tuberculosis.

The liver is slightly enlarged. The greater part of the surface is smooth and yellow. The right anterolateral margin is reddened and firmer than the adjacent tissue for a distance of 3 or 4 cm. from the edge. On the anterior surface within this area are two small elevations, each about 1.5 cm. in diameter, which are yellow and fluctuating. Scattered over the remainder of the superior surface are a few, small, reddish, non-elevated areas which are softer than the sur-

rounding tissue. The greater part of the left lobe is reddened and increased in consistence. On the inferior surface, near the tip, is a small, soft, yellow elevation, and extending from this toward the hilum of the organ is an elevated, mottled, yellowish-red ridge. On the superior surface is another small, yellow elevation to which a little fibrin is adherent.

FIG. 3.—Liver abscesses resulting from ascarides in bile ducts.

On opening the duodenum about two-thirds of a long *ascaris* is seen projecting from the orifice of the common bile duct. The orifice is closed by the worm, but the duct above it is markedly dilated and contains parts of other *ascarides* which extend up into the liver. The mucosa of the duct is smooth but reddened. Both the right and left branches of the hepatic duct are dilated and filled with *ascarides*, those in the left branch causing the ridge seen on the surface of the left lobe.

The yellow areas on the surface overlie small abscess cavities which connect with the bile ducts, but are closed, or partially closed, by *ascarides*. The parenchyma in the reddened areas surrounding the abscesses and dilated bile ducts is moist and friable; elsewhere it is soft, yellow and greasy.

The worms are not confined to the main branches, but extend into

the smaller ducts and into the abscesses. The total number of parasites present is between fifteen and twenty, it being impossible to count them without removing them from the liver. The majority of the worms are well preserved and apparently alive, though motionless; but on both the right and left side are found one or two which are dead, macerated and bile-stained. The largest measures about 18 cm. in length.

The cystic duct is slightly dilated. The gall-bladder is not enlarged and contains turbid, light green bile. Its mucosa is bile-stained and not reddened. Three *ascarides* in stomach and seventy-eight in intestine.

2387. *Ascarides* in bile ducts; cholelithiasis; cholangitis; cholecystitis; abscesses of liver.

Male, Filipino, aged forty years. Duration of illness, two days. Autopsy performed seventeen hours after death.

The gall-bladder is enormously distended, projects about 5 cm. beyond the costal margin and measures in its distended state about 10 cm. in its lateral diameter. On palpation of the common duct a firm mass is felt in its lower portion. On opening up the common duct an opening is found in the duodenum, which is considerably larger than the normal opening of the duct, and on closer examination the true orifice is found about 1.5 cm. from this, the first opening representing a fistula. About 1 cm. back of this fistulous opening are two brownish stones, one large (1.2 cm.) and the other small; these have not entirely obstructed the duct, however, since bile could be pressed from the gall-bladder before the duct was opened. Just back of these stones lies a brownish, coiled-up mass which is found to be an extremely long *ascaris*, which is flattened and covered with deposits of bile pigment. The mucosa of the duct at the point where the stones lay is hyperemic and shows two or three small greenish-colored, shallow ulcers; back of this point it is markedly dilated as is the hepatic duct and its branches. No other stones are found except a small one in a branch of the hepatic duct. The gall-bladder contains a thin, rather sticky fluid, which is fairly clear but contains occasional yellowish or brownish particles. The mucosa of the gall-bladder is intensely hyperemic, and scattered over it are numerous shallow ulcers, which are stained a deep green color, so that at first they seem to be small patches of membrane.

The liver is large; on the surface are seen several yellowish dots, most numerous on the left lobe. On section of the right lobe the ducts are seen to be greatly dilated and in one of them, and apparently attached to its wall, lies an *ascaris;* the worm is in a good state of preservation, though apparently dead at this time; near it in the duct is another worm. Scattered here and there through the liver substance are small, yellowish areas, 2 to 4 mm. in diameter, and in the vicinity of many of these there is evidently an increase of connective tissue; these areas are infrequent in the right lobe, but

on section of the left lobe are found more often. The remaining liver parenchyma is reddish brown and rather friable. No *ascarides* were found in the intestine or stomach.

2524. *Ascarides* in bile ducts, gall-bladder and intestine; abscesses of liver.

Male, Filipino, aged five years. Autopsy two hours after death. Died of tuberculosis.

The liver, about normal in size, is firm and of a yellowish color. On the diaphragmatic surface are seen four well-defined abscesses of a yellowish-green color, averaging about 1 cm. in diameter, fluctuating on pressure. On the inferior surface of the right lobe is a linear abscess about 2 cm. long and 0.5 cm. wide, of a greenish color. None of these abscesses has penetrated the capsule. The organ cuts with decreased resistance; the cut surface is yellowish and dry. The lobulations are not distinct. The abscess cavities are well defined and apparently communicate with the larger bile passages, which contain living and much disintegrated dead *ascarides*. The liver weighs 575 grams.

The gall-bladder is much distended with green bile and contains two large living *ascarides*. The mucosa shows no gross change. Many *ascarides* in intestine.

2797. *Ascarides* in bile ducts, small intestine and stomach; abscesses of liver.

Male, Filipino, aged one year and seven months. Interval between autopsy and death unknown. Died of Asiatic cholera.

The left lobe of the liver looks red and inflamed. Upon section this portion is found to contain a dead *ascaris* within a bile duct, which is dilated and contains a small amount of yellow fluid. Around this duct are seen many small pale areas, irregular in outline, about 1 to 5 mm. in diameter; some are larger. These areas are soft and structureless in the centers. Around the periphery the liver tissue is red and swollen. There is another *ascaris*, which is alive in a branch of the hepatic duct. The surface of the liver is smooth. The organ is soft. It presents a dull, gray, mottled appearance. Many *ascarides* are present in the small intestine and stomach.

1007. *Ascarides* in bile ducts, liver, intestine, stomach and esophagus; abscesses of liver.

Male, Filipino, aged five years. Duration of illness, one month. Autopsy performed on day of death.

Multiple abscesses in liver up to 4 cm. in diameter. One large abscess on dome of right lobe, its upper wall formed by the diaphragm. Abscesses contain greenish, foul-smelling pus and *ascarides*. Common bile duct, hepatic duct and intrahepatic bile ducts and abscesses filled with *ascarides*. General suppurative peritonitis apparently originated from the subdiaphragmatic abscess. Base of the right lung was adherent to the diaphragm and there is here localized empyema and gangrene of the lung. Gall-bladder free. *Ascarides* also present in ileum, cecum, stomach and esophagus.

1929. *Ascarides* in bile ducts, stomach and intestine; fluctuating projections on the surface of the liver. Case of sudden death in a subject of status lymphaticus.

Male, Filipino, aged five years. Duration of illness, thirty minutes. Autopsy performed the day after death.

Liver weighs 720 grams. Common bile duct, hepatic and intrahepatic ducts are much dilated and contain numerous *ascarides*. These extend from hilum to capsule. Fluctuating projections on the surface of the liver, but no actual abscesses described. Ten to twelve *ascarides* in stomach and numerous in intestine. Base of right lung adherent to diaphragm.

3192. *Ascarides* in bile duct, pancreatic duct and intestine; bile duct dilated.

Male, Filipino, aged forty-two years. Duration of illness, two days. Autopsy performed sixteen hours after death. Died from transverse myelitis and fracture of the seventh cervical vertebra.

In the hepatic and common duct is found a dead *ascaris* folded on itself; one end of it macerated and bile-stained. The bile duct is slightly dilated, but otherwise is apparently normal. A large *ascaris* has penetrated the pancreatic duct to a depth of about 8 cm., the worm projecting into the duodenum. The worm is still living. No lesions of the pancreas are made out. A few *ascarides* are present in the intestine.

3905. *Ascarides* in bile ducts and intestine; abscesses of liver and pancreas; cholelithiasis; icterus; hemorrhages.

Male, Filipino, aged seventeen years. Duration of ilness, four days. Autopsy performed twelve hours after death.

Jaundice is present. There is hemorrhage into the peritoneal sac without recognizable source of origin. There is also abundant, altered blood in the intestine, stomach, mouth and nose, which may have originated from the necrotic ampulla of Vater. Extensive hemorrhages are present in the retroperitoneal tissues, about the kidneys, in the pelvis, in the subserous coat of the jejunum and in the subpleural tissues. The liver is enlarged and weighs 1661 grams. It shows many small abscesses from 3 to 8 mm. in diameter, containing green pus. The common bile duct is 4.5 cm. in circumference and contains nine greenish black facetted smooth stones. Four dead and decomposed *ascarides* are found in the common and hepatic ducts and their branches. There is also much pus in the pancreas, especially near its tail. Its ducts are dilated but do not contain bile. The gall-bladder is dilated and contains dark green bile. There are forty-two living *ascarides* in the intestine.

3579. *Ascarides* in bile ducts, gall-bladder and intestine; possible postmortem migration.

Female, Filipino, aged five years. Duration of illness, fourteen days. Autopsy performed eighteen hours after death. Died of Asiatic cholera.

Liver is about normal in size. Located in the right hepatic duct, entering into the substance of the liver for a distance of about 4 cm., there is an *ascaris*. This *ascaris* is curled upon itself in the hilus of the liver and the other half enters into the left main hepatic duct for a distance of about 2 or 3 cm. Located in the gall-bladder and common bile duct there is an *ascaris*. The gall-bladder contains, besides the *ascaris*, a small amount of thin, turbid, greenish bile. "*Ascarides* are present" in the intestine.

2896. *Ascaris* in cystic duct; dilatation of gall-bladder.

Male, Filipino, aged four years and one month. Autopsy performed nine hours after death. Died of acute malaria.

Gall-bladder is distended, forming a large, sausage-shaped, cystic mass, with thin wall. It contains some golden yellow, very thin bile. A living *ascaris* is present in the cystic duct, its head extending up into the gall-bladder, and its tail is just at the point of junction of the cystic and common ducts. The common duct is not dilated. No mention is made of other *ascarides* in the intestine.

MIGRATION TO THE PANCREAS. Worms may pass into the duct of Wirsung and determine either the formation of abscesses or the development of an acute hemorrhagic pancreatitis. It also seems probable that the *ascaris* in the common bile duct may so block it as to lead to the development of an acute hemorrhagic pancreatitis without the entrance of the worm into the duct of Wirsung.

CASES OF ASCARIS IN PANCREAS.

Cases 4424 and 3905 have been already described along with the cases of *ascaris* in the bile ducts and liver.

4582. *Ascarides* in pancreatic duct and intestine.

Female, Filipino, aged forty years. Duration of illness, one year. Autopsy performed sixteen hours after death. Died of tuberculous pneumonia.

The only pertinent note is that an *ascaris* was found in the pancreatic duct and there is a slight hemorrhagic area at the tail of that organ. Numerous *ascarides* in the intestine.

2478. *Ascaris* in pancreatic duct and intestine; probable postmorten migration.

Female, Filipino, aged seventeen years. Duration of illness, thirteen days. Autopsy performed sixteen hours after death. Death from acute endocarditis and myocarditis.

The only pertinent note is that the pancreas shows a large *ascaris* well engaged in the duct of Wirsung. Pancreas shows no change. Few *ascarides* in the intestine.

MIGRATIONS OF THE ASCARIS TO OTHER PARTS. The migration of the *ascaris* to the stomach and esophagus and thence to expulsion through the mouth or nose is extremely frequent. If fistulous tracts are encountered *en route* the worm may reach unusual places.

It may also pass into the accessory nasal sinuses, antrum of Highmore, lacrimal duct, Eustachian tube and external ear or into the larynx and trachea. In one of our cases a live *ascaris* was found in the middle ear of a child after death.

Passage of the worm into the larynx may cause suffocation, and numerous cases are quoted by the authors already referred to in which removal of the worm from the larynx or trachea had led to the disappearance of alarming symptoms. The precaution has already been suggested of hesitating to ascribe harmful effects to the presence of *ascarides* found in the respiratory passages after death unless signs of death by asphyxia are present.

Case 5684 described above illustrates the effects of *ascaris* in the respiratory passages.

II. The Ascaris a Carrier of Infection in its Migrations. This subject needs only to be appreciated. The way in which infection has been carried to the peritoneum, liver, gall-bladder and pancreas, giving rise to suppurative inflammation in these places, has already been exemplified in the above cases and constitutes one of the serious dangers of ascariasis.

III. Bronchopneumonia in the Course of Ascaris Infection. Stewart[8] and Ransom and Foster,[9] in their studies on the life history of *ascaris* in experimental animals, have found that a bronchopneumonia may develop and may prove fatal, if the infection is massive, from the presence of the larvæ in the lungs of these animals (pig, rat, mouse and guinea-pig). In a case of human experimental infection, Lutz[10] recorded the occurrence of a severe bronchopneumonia in the subject of the experiment during the incubation period. From this it seems not improbable that some, at least, of the cases of bronchopneumonia in infants and children may be due to the presence of the larvæ in the lunge, and thus constitute a hitherto unsuspected danger of *ascaris* infestation.

IV. Toxic and Reflex Nervous Symptoms from Ascaris. In the present state of our knowledge it is difficult to speak with any degree of accuracy upon this important and interesting phase of ascariasis. I have grouped together the toxic and reflex nervous effects for purposes of discussion because of their frequent association and the practical difficulty in distinguishing between them. It is probably true that the number of cases of ascariasis which come under this head is immeasurably greater than those which come into the hands of the pathologist, which have been described above. Clinical histories and the therapeutic effects of vermifuges unite to incriminate the *ascaris* as the cause of almost all sorts of toxic and nervous phenomena. Fevers, nausea, flatulence, abdominal pains, convulsions, tetany, symptoms of chorea, hysteria and epilepsy,

[8] British Med. Jour., 1919, p. 102.
[9] Jour. Agric. Research, 1917, xi, 395.
[10] Quoted by Stewart.

delusions, hallucinations, psychic disturbances, symptoms simulating meningitis and many other disorders of like nature, which occur in the course of ascariasis and disappear after successful treatment of the ascariasis, come within the ken of those who practice medicine where the *ascaris* abounds. It is therefore logical to ascribe an etiological role to the *ascaris* in these conditions, but the way in which it produces its results is by no means clear.

Flury[11] in a chemical and toxicological study of the horse- and pig-ascaris found numerous substances both in the body substance and in the excretions which caused local irritation or necrosis. He was able to demonstrate volatile aldehydes of fatty acids and even the free acids, the principal ones being valerianic and butyric acids, and in smaller amounts formic, acrylic and propionic acids, alcohols and esters of the ethyl, butyl and amyl series. All of the symptoms of irritation of the mucous membranes noted by zoölogists and the digestive disturbances in ascariasis he attributed to these substances. All of the disturbances of the central nervous system attributed to *ascaris* (hallucination, hysteria, chorea, epilepsy, cramps, tetanus, delusions, psychic disturbances) may be explained on the basis of chronic aldehyde poisoning. A nitrogenous compound was found which caused death, with severe hemorrhages in the intestine after subcutaneous injection in the dog. This was ascribed to its action as a capillary poison. Flury concludes that the *ascaris* produces not a single poison but a number of pharmacologically active substances which may produce very different and at times very severe symptoms.

Shimamura and Fujii[12] have isolated a toxic substance from the horse *ascaris* to which they have given the name *askaron*. This consists of a mixture of albumoses and peptone and gives distinctly toxic symptoms when injected into the horse subcutaneously or intravenously. It is said that this *askaron* is not a specific toxin produced for a definite purpose, but is merely a metabolic substance that happens to be toxic.

Cattaneo[13] was able to detect only a weak toxin in *ascaris*, while Messineo,[14] by injecting into animals extracts in physiological salt solution, invariably succeeded in producing serious motor disturbances and frequently death.

Whether other toxic effects than those enumerated above can with any degree of probability be ascribed to the *ascaris* is a matter of debate. The pathologist sees many cases of disturbances of the spleen, and to a lesser extent of the liver, which are not accounted for by any of the known etiological agents, and he cannot help wondering whether the *ascaris* by itself or by some change caused by it in the intestine may not play an important role in this respect.

[11] Arch. f. exp. Path. u. Pharm., 1912, lxvii, 275.
[12] Saikingaku Zassi, 1916, p. 9.
[13] Arch. f. Kinderheilk., vol. xliv. Quoted by Fantham, Stephens and Theobald.
[14] Giorn. Med. del regio eserc., 1905. Quoted by Fantham, Stephens and Theobald.

CASES OF ASCARIASIS AND HEMORRHAGES.

3905. See above with "*Ascaris* in Bile Ducts and Liver."

2029. Already published in full by Crowell and Hammack. Briefly, this was an eight-year-old Filipino, who, six days before death, was seized with an attack of vomiting, followed on the next day by pain in the chest and knees. There soon developed external edema, jaundice, melena, hemorrhage from the gums, dyspnea, prostration and ecchymoses on the face and legs. The day before death there were severe anemia, hemic cardiac murmurs, negative Widal reaction, sterile blood culture and no fever. The anatomical diagnosis at autopsy was: ascariasis (150 *ascarides* in stomach and intestine); purpura hemorrhagica; ecchymoses, cutaneous, epicardial, pleural, retropleural, retroperitoneal, gastric and intestinal; partial atelectasis of lungs; anemia; hemorrhage into intestines; trichuriasis; ankylostomiasis.

CONCLUSION. The dangers of ascariasis are liable to be underestimated, and for this reason a number of cases, fatal and otherwise, have been here reported for the purpose of attracting attention to this important subject. These worms may cause symptoms and even death through toxic, reflex and mechanical effects either in the larval stage or while adult in the intestine or in the course of their migrations to other parts of the body. Cases of disease of the intestine or other parts may assume much more than their usual seriousness on account of the presence of these worms, and the accidents due to the migratory proclivities of the worm may cause fatal complications of simple surgical procedures that would otherwise have terminated happily. The number of cases that I have here recorded, which constitutes only a fraction of those I have seen, is not great. It, however, indicates that a thorough and systematic examination of the cases of ascariasis would bring to light as many cases in the experiences of others, and would prove ascariasis to have the importance that I have ascribed to it.

EVIDENCES OF NEPHRITIS AND URINARY ACIDOSIS.

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IF the reader of this discussion finds that he cannot subscribe to the title under which I shall treat the present subject, I will beg his indulgence on three points: (1) on the fact that if a specimen of urine were submitted to him for analysis and he found it to contain albumin, blood cells and casts, he could not possibly deny that the individual from whom the specimen was obtained had a neph-

ritis; (2) that such findings are the main evidences of an actual nephritis in all but the late stages of disease; (3) that if such findings occurred in the course of an acute infection, for example, the inference would almost invariably be that a nephritis existed.

I therefore prefer to treat the subject-matter of this paper from the viewpoint of nephritis, because, from such a viewpoint these findings have greater clinical significance, and not because I believe that a true nephritis existed. And also because the acidosis theory of nephritis has up to the present time been neither accepted nor rejected.

Previous Observations. In an earlier work[1] it was shown that severe and prolonged muscular exertion is accompanied by circulatory disturbances sufficient to cause the presence of blood cells, albumin and casts in the urine. Those observations suggested the inquiry whether anything short of a Marathon race (24.85 miles) would produce similar findings, and the question is answered in the present investigation.

It is interesting to note that an individual who immediately before exercise had a perfectly normal urine will, after forty minutes of exercise, show albumin, red blood cells, hyaline and granular casts. It is indeed surprising to note the rapidity with which hyaline and granular casts are formed in the urinary tubules.

Before going further, I wish to quote a summary of the observations made in Marathon runners:

URINARY FINDINGS BEFORE MARATHON RACE, IMMEDIATELY AFTER AND AT SUBSEQUENT PERIODS.

	Number of cases.	Albumin.	Blood.	Casts.	Acet. bodies.
Before	24	1			
Immediately after	19	19	18	19	18
One week later	19	19	..	6	
Three weeks later	3	3	..	3	

As will be seen from the above table this inordinate exertion, running a distance of 24.85 miles in a period of 3 hours 14 minutes to 4 hours 15 minutes, produced albuminuria accompanied by blood and casts in all the cases. Five of these showed "showers of casts."

We made also numerous observations on the blood-pressures of those subjects, and in correlating the findings it became evident that albumin occurred in largest amounts in those cases which showed the greatest fall in the maximum pressure and in those which showed most marked falls in the pulse-pressure. Those findings correspond with the studies of Erlanger and Hooker,[2]

[1] Barach, Joseph H.: Physiological and Pathological Effects of Severe Exertion (the Marathon Race) on the Circulatory and Renal Systems, Arch. Int. Med., 1910, v, 382.

[2] An Experimented Study of Blood-pressure and Pulse-pressure on Man, Johns Hopkins Hosp. Rep., 1904, xii, 145.

who showed that a direct relationship exists between pulse-pressure and urinary excretion. Later on it was shown by Gesell[3] that in the excised kidney, with a given amount of blood, the urinary excretion is greater when the pulse-pressure is larger, diuresis being determined by the size of the pulse-pressure.

The conclusions derived from that study were: From these observations upon the renal functions it seems that the more serious the disturbance of the general circulatory system the more marked are the evidences of this disturbance in the renal circulation. This is shown by the amount of blood and the degree of albuminuria and cylindruria. In these observations no quantitative analyses of the urinary acidity were made.

Since that work, however, it has been stated by Bornstein and Lippman,[4] who are in agreement with Martin Fisher, that the albuminuria and cylindruria following severe exertion is caused by the increased urinary acidity;[5] the acid products of increased metabolism irritating the kidneys. This is a most important question in clinical medicine, and we may well bring all of our evidences together for the proper solution of such a problem.

PRESENT STUDY. The following observations were made upon a series of 57 normal young men before and after baseball and daily "try-outs" on the track. These young men were of better than average health, having passed the required physical examination prior to their entrance into the field of competitive sports.

Our observations included the body weight before and after effort, urinalyses before and immediately after, including the amount of urine excreted during the work period, estimation of the hydrogen ion concentration of the urine and examination for albumin, casts and blood cells.

LOSS OF WEIGHT AND URINARY EXCRETION. In the entire series the greatest loss of weight occurred in a subject who weighed 129½ pounds. His loss in 1 hour and 50 minutes of baseball was 5.5 pounds. One subject showed no loss at all and the average loss for all cases was 1.3 pounds. This decrease in body weight was mostly due to loss of water.

The 10 individuals showing the largest amount of urine, and whose average excretion was 87 c.c., lost on an average of 1.55 pounds each, while the 10 whose average was 27 c.c. lost on an average 1.1 pounds each.

DURATION OF THE EFFORT. The average time spent in running was 48.1 minutes. The average time spent in baseball was 1 hour 57 minutes.

[3] The Relation of Pulse-pressure to Renal Secretion, Am. Jour. Physiol., 1913, xxxii, 70.

[4] Weitere Beiträge zur nicht Nephritischen Albuminurie, Ztschr. f. klin. Med., 1918, lxxxvi, 345.

[5] Gesell, R.: Loc. cit.

Taking the first 10 who spent the longest time and the 10 who spent the least time in the exertion the number in which albumin, blood cells and casts were found was exactly the same in both, showing that in this series time element was not a factor.

URINARY ACIDITY. In order to estimate the hydrogen ion concentration, *i. e.*, to estimate the total potential acidity of the urine, we used the method of Folin, which consists of adding a liberal quantity of neutral potassium oxalate to the urine, and then titrating with $\frac{N}{10}$ NaOH, using phenolphthalein as an indicator.

By this method we found that 85 per cent. of all the urines after exercise showed an increased acidity. The actual number of increased acid urines was exactly the same in the baseball cases as in the more strenuous type—the track cases.

The acidity was greater in the 10 cases which showed an average urinary output of 27 c.c. than in the 10 which had an average output of 87 c.c. during the exercise period.

Albumin and casts occurred as frequently in the low acid cases as in the high acid cases.

The average increase in total acidity for the cases showing casts was 16.8—as against an acidity of 18.8 in cases not showing casts.

From all this, the deduction which seems warranted is that the height of the urinary acidity does not determine the presence of albumin, blood cells and casts.

ALBUMINURIA. Seventy-seven per cent. of the entire series showed albuminuria as a result of the effort.

Out of 36 "baseball" cases, 26 showed albumin and 10 showed none.

Out of 18 "track" cases, 16 showed albumin and 2 showed none.

The inference here is plain that albuminuria occurs more frequently as a result of strenuous effort, *i. e.*, those cases in which there is the greatest amount of circulatory disturbance.

MICROSCOPIC FINDINGS. *Casts and Red Blood.* After running on the track 71 per cent. had hyaline and granular casts and a small percentage of red blood cells. After baseball 23 per cent. had hyaline and granular casts or red blood cells. From this we may also infer that the more strenuous type of exertion causes casts and blood cells to appear in the urine.

RÉSUMÉ. These observations show that in the most severe type of exertion (Marathon race), albumin and casts and red blood cells occur in all individuals. The casts were of the broad and narrow, hyaline and granular variety. Some of the casts showed red and white corpuscles. The amount of albumin was greatest in those who ran most strenuously, finishing earliest, 3 hours 14 minutes to 4 hours 15 minutes.

Observations of the blood-pressures and pulse-rate in these cases showed that the largest amount of albumin and blood and casts occurred in those individuals who showed the greatest degree of

circulatory disturbance. This was manifested by a marked fall in both the maximum blood-pressure and in the pulse-pressure.

In the less strenuous type of exertion we usually find an increased urinary acidity, albuminuria, cylindruria, and at times blood cells.

A critical analysis of these findings, however, shows, first of all, that in the more strenuous effort the amount of albumin, the presence of blood cells and the number and type of casts all depict a more serious renal disturbance than is found in the less strenuous form of exercise.

Urinary acidity was increased after exercise in 85 per cent. of the cases, but it did not occur more frequently, nor was the acidity higher in the severe exertion cases than in the milder ones.

There is a positive relationship between the degree of albuminuria and cylindruria and the type of physical exertion; but these observations show that no such relationship can be established between the urinary acidity and the occurrence of albumin, casts and blood cells in the urine.

THE ASSOCIATION OF FEVER WITH FRACTURE OF THE SKULL.

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A slight rise of temperature is so common after fracture of the skull—with and without injury of the intracranial contents—that one rather expects it in almost every case; the rise is rarely above 100° F., is short-lived and lasts for approximately twenty-four hours, after which subsidence to the normal occurs and the latter continues until convalescence is completed. In some of the patients, however, fever assumes a more important role: it rises to extraordinary heights; it persists for comparatively long periods of time; it indicates some grave complication or the reaction to an extensive trauma. The cases in this latter group form the basis for this communication.

Such fever occurred in 15 of a series of 72 cases of fracture of the skull which were recently studied—21 per cent. In another series of cases of fracture of the skull, admitted to the hospital in a different period of time, 22 patients of a total of 77 developed fever—29 per cent. In the 15 febrile cases of the first series the fractures were situated in the posterior fossa in 6, and in the middle fossa in 2; in the others the fractures were distributed over the vertex and sides of the skull. One fracture was compound externally; one communicated with the middle ear and 2 with the nasal cavities; the

others were all closed fractures. Eight of the patients who developed fever died; in 4 of these the cause of death was a meningitis.

The cases which developed fever can be divided into the following groups:

1. The Ordinary Cases:

In a number of the febrile cases the temperature was of a moderate degree and the cause for the latter could not be established. In one of the cases (No. 192204) fever was present for two days. Except for the fact that it persisted somewhat longer than usual it approximated to the initial reaction so frequently seen and would, ordinarily, draw no extra comment. In another patient (No. 156919) there was an initial rise of temperature which fell promptly as it usually does; on the fourth day it rose again to a moderate height to fall to the normal after twenty-four hours more. Both of these patients recovered and the cause of the temperature remains obscure.

Case No. 192204. A boy, aged seven years, fell from a fire-escape a distance of thirty feet and landed on the front and side of his head. For a period of thirty minutes he was unconscious; thereafter he vomited a number of times and complained of pain in the left side of his head. The physical examination showed a hematoma of the scalp and the roentgen ray showed a frontoparietal fracture. The neurological status was normal except for some hyperactivity of the knee-jerks. The character of the fever is indicated in Fig. 1. The patient made an uninterrupted recovery.

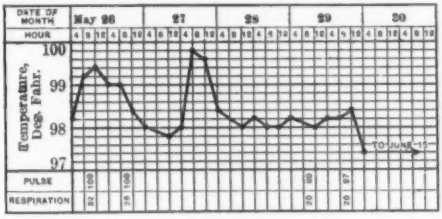

FIG. 1

Case No. 156919. A boy, aged seven years, fell a distance of eight feet and struck the right side of his head. He was unduly quiet thereafter and complained of a dull headache; he vomited after taking some food. There was a hematoma of the right parietal region and the roentgen ray demonstrated that the fracture lay directly underneath and extended into the mastoid. There were no neurological disturbances. The fever is shown in Fig. 2. An uninterrupted recovery followed.

2. The Fatal Cases:

In the following case an excessive degree of fever was associated with an extensive cranial fracture and with widespread disorganization of the intracranial contents. The notes of the case follow.

Case No. 179660. A six-year-old girl who was thrown from an automobile struck the pavement with the top and back of her

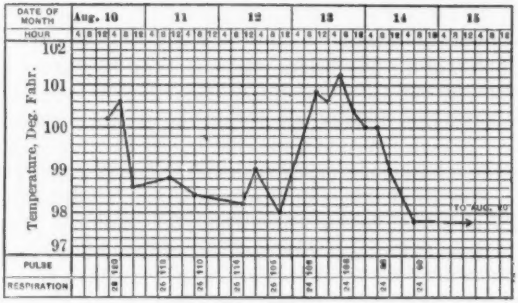

FIG. 2

head. There were several large hematomata over the occiput and vertex and behind the ear. The neurological disturbances which

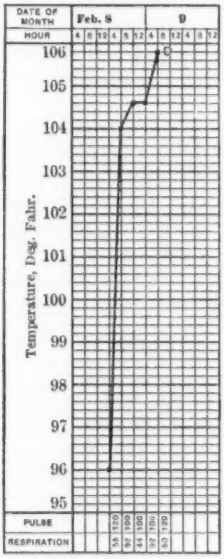

FIG. 3

pointed to a brain injury included (1) unequally dilated pupils, neither of which reacted; (2) internal rectus paralysis; (3) beginning

papilledema; (4) spasticity of all four extremities, with absent abdominal and exaggerated limb reflexes and elicitable abnormal reflexes, including Babinski, Gordon and Oppenheim responses; (5) peculiar athetoid movements of the hands, with flexion of the hands at the wrists; (6) stupor, with Cheyne-Stokes breathing. At the time of death—about twenty hours after the injury—there was (7) complete flaccidity, with (8) absent reflexes and (9) widely dilated pupils. Prior to death the temperature reached 106.8° F. (Fig. 3).

The cause of death in this child was evidently a widespread disorganization of brain substance, aided by a marked grade of intracranial compression from edema and hemorrhage. The temperature, owing to the shortness of time elapsing between the injury and death, assumes the characteristics of an antemortem rise. Our knowledge in this regard is, however, very deficient, and it is quite probable that this as well as other antemortem rises of temperature have something in common with disturbances of the heat-regulating centers.

The notes of two other fatal cases are given in which fever formed one of the prominent parts of the symptom-complex.

Case No. 190666. A fifty-five-year-old patient sustained a bad crushing fracture of the right malar region and side of the skull immediately opposed to it; there was very marked protrusion of the orbital contents. Besides the last the clinical picture included much shock; marked restlessness; fever (Fig. 4); a sluggish,

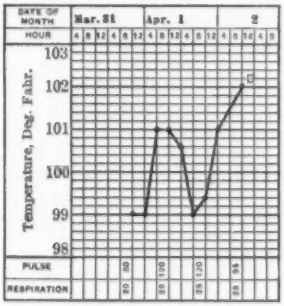

FIG. 4

irritable and disturbed mentality, which changed to stupor, deepened to coma, and terminated in death forty-eight hours after the injury was sustained. The character of the symptoms, taken in conjunction with the location of the fracture, pointed to disturbances in the frontal lobe.

Case No. 173080. In the second of these two patients, a woman, aged sixty years, a fracture of the base of the skull was associated,

clinically, with rigidity of the neck, coma, general spasticity and loss of control of the sphincters. Death occurred seventy-two hours after the injury. Fever (Fig. 5) was present during the entire period the patient was in the hospital.

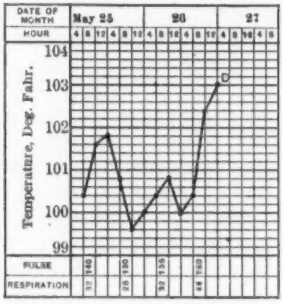

FIG. 5

In both of these patients the probabilities are very strong that there was considerable intracranial hemorrhage; in the second patient the symptoms indicated cerebral compression; in neither of them was there any indication present of any infectious process or of any complicating condition which could account for the irregular temperature.

3. Fever in Compound Fractures:

In one patient fever, as noted in Fig. 6 (Case No. 185032), was associated with a compound fracture. The scalp wound had been

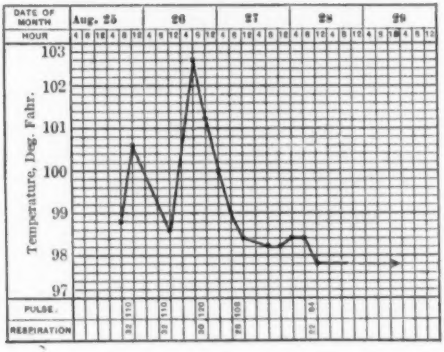

FIG. 6

sutured immediately and, apparently, had healed by primary intention. In the absence of any other cause it is quite likely, however, that infection was present in the wound and was combated

by the natural powers of the body without any disturbance of the healing.

A very remarkable case is the following:

Case No. 193199. The patient—a child, aged six years—sustained a fracture in the posterior fossa of the skull. Nothing extraordinary was noted at the time of admission to the hospital, and there were no signs of disturbed neurological function. Fever of a slight grade (Fig. 7) was present from the beginning, and by the fifth day it had risen to 102.4° F. During the course of the routine examination a laceration was discovered in one drum-head, and owing to the positive history of no preceding affection of the ear the otologist (Dr. Braun) rendered the opinion that the fracture had opened into the middle ear and, presumably, had become secondarily infected. The otitis subsided very quickly, however, and ten days after the injury the perforation in the drum-head had closed completely. On the thirteenth day the temperature reached 104° F. Lumbar puncture had hitherto not been practised, owing to the recent work of Weed and his co-workers, and of Wegeforth and Latham, but at this point it was done, and was subsequently repeated two days later; except for some increase of pressure on the first tapping the procedure yielded negative information both times: there were 3 cells per cubic millimeter and the fluid was bacteriologically sterile.

During the second week the following neurological signs developed rather slowly and irregularly: (1) A weakness of one facial nerve; (2) a convergent squint; (3) slight twitching of the left upper extremity; (4) very slight retraction of the head, with moderate rigidity of the neck which could, however, be forcibly overcome; (5) tendency to a Kernig on the right side; (6) irritability. No abnormality could be discovered in the chest or abdomen and no focus of infection could be demonstrated in any of the limbs. In the third week the temperature gradually subsided to normal, the signs and symptoms all gradually disappeared and thereafter the child made a perfect recovery.

The question in this particular case was whether the child had had a basilar meningitis, from which it had recovered, or whether the fever was due to some other cause. The probabilities of a meningitis were much strengthened by the fact that the patient was admitted into the hospital a short time after the termination of the influenza epidemic; coincident with the stay of this patient in the hospital another child had been admitted to the same ward and had gone through a similar clinical picture—in which similar negative laboratory findings were demonstrated—and, after it had died, an autopsy revealed a basilar meningitis, with a shaggy, greenish exudate in which influenza-like organisms were demonstrable in smears. The physical findings were in no way conclusive, inasmuch as cases of basilar or posterior fossa fractures quite

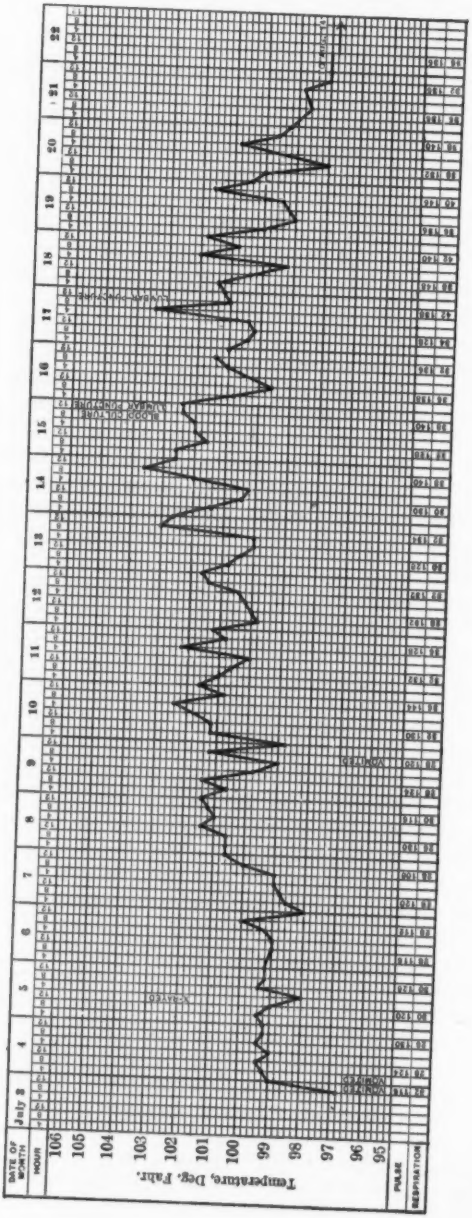
Temperature, Deg. Fahr.
106
105
104
103
102
101
100
99
98
97
96
95
DATE OF MONTH
July 3
HOUR
PULSE
RESPIRATION

FIG. 7

commonly exhibit some of the signs of meningeal irritation. The probabilities, when all things are considered, seem to be that the physical findings were due to irritation of the meninges by the extravasation of blood and that the fever was due to absorption of the latter and to the aseptic inflammatory processes necessary to the proper repair of the fracture.

4. Fever Due to Complications:

Fever occurred also as a reflection of some complicating condition. The most prominent of these was a meningitis. The two cases given as examples show the varieties of fever which were present when this complication appeared after fracture of the skull; in the one (No. 177900) the infection was fulminant, the fever was high and the clinical course was very short and terminated in a fatality; in the other (No. 173371) the symptoms and fever made their appearance after several days and the course was more protracted.

Case No. 177900. The patient had a fracture in the frontal region and the roentgen ray showed, clearly, the communication with the frontonasal sinuses. The signs of meningitis were developed fully within twenty-four hours and the spinal fluid was loaded with polymorphonuclear cells and contained type I pneumococci. Fig. 8 shows the character of the fever.

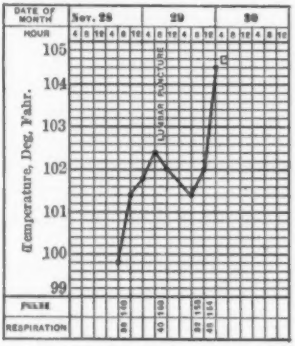

FIG. 8

Case No. 173371. In falling a five-year-old lad struck the pavement with the back of his head. He vomited and became unconscious; later this disappeared. The eyeballs showed some nystagmus when turned to the right, otherwise the neurological status was normal. Three days later the temperature rose to 105° F. The patient became apathetic; he assumed an attittude of complete flexion; there was moderate opisthotonos, a bilateral Kernig, and a suggestion of a Babinski. The spinal fluid contained many cells and Type IV pneumococci. The patient died.

The postmortem examination showed a fracture extending to the left jugular bulb and into the left middle ear and a general purulent meningitis.

Fig. 9 shows the temperature curve.

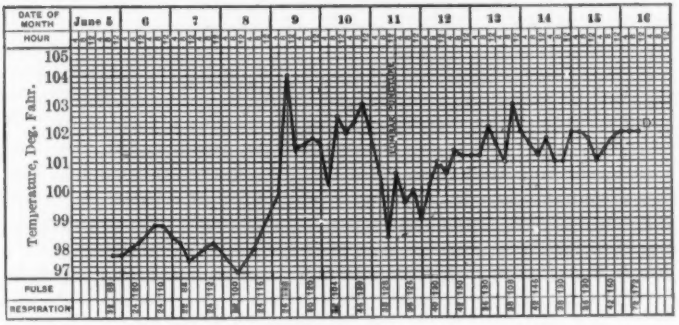

FIG. 9

5. Fever in the Operated Cases:

Fever in the operated cases was present in some both before and after operation and in others only after operation. In the latter it was frequently so because the patients were operated upon immediately after admission to the hospital and there was no opportunity allowed before the interference for the temperature to rise. When excessive fever developed after operation there was never any conclusive proof that it was directly due to the operative intervention, and, in all probability, the nature of the injury and of the resultant pathological changes contributed largely, if not entirely, to the pyrexia. The following cases are good examples of those in this group.

Case No. 161336. One hour before admission the patient fell down an elevator shaft. The patient was pale, semiconscious and in marked shock. In the parietotemporoöccipital region was a large hematoma. Blood was escaping from the ear. There was a progressive lowering of the pulse-rate to forty-four beats to the minute. The neurological status included (1) an initial period in which the reflexes were absent; (2) periods of excitation and stupor alternating with one another; (3) paralysis of left arm and leg; (4) reappearance of the reflexes which became hyperactive; (5) clonus at both ankles.

The operation, which was done four hours after the injury, showed a fracture extending from the occiput into the temporal bone and mastoid and which was depressed in areas. The dura was plum-colored and there was a perforation in the lateral sinus. An extensive subdural hemorrhage was also present and the brain

was badly lacerated. Bilateral subtemporal decompression was done.

On the following day improvement was present in that power began to return in the arm. Twenty-four hours later, however, the patient's condition became worse and death followed shortly. The character of the fever is shown in Fig. 10.

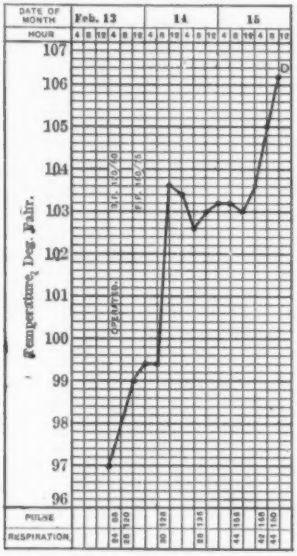

FIG. 10

Case No. 148405. A boy, aged nine years, was run over on the street and was brought immediately thereafter to the hospital. On admission the boy was in stupor and was bleeding from the nose and mouth. The neurological findings included (1) left facial weakness; (2) twitchings of left upper limb; (3) convulsions beginning in the left arm, spreading to the left leg and to the opposite side of the body. Within the next twenty-four hours the signs of compression grew more marked.

Operation[1] was done on the day following the injury. A flap operation was done. The exploration showed (1) that no hemorrhage was present; (2) great tension and edema of the brain; (3) some bloody serum in the basilar portion. No improvement followed the operation and the patient died approximately twenty-four hours later.

[1] The reasons for the operation and for the exploration through a bone flap are not apparent from the history.

In Case No. 148405 fever (Fig. 11) was present from the time of admission to the hospital and reached 102.8° F. within twelve hours. At the time of operation—forty-eight hours after the injury—the temperature had reached 106.6° F. There was marked hyperpyrexia at the time of death. The injury included a basilar fracture and much disorganization of brain tissue, as evidenced by the great tension and widespread edema of the cortex.

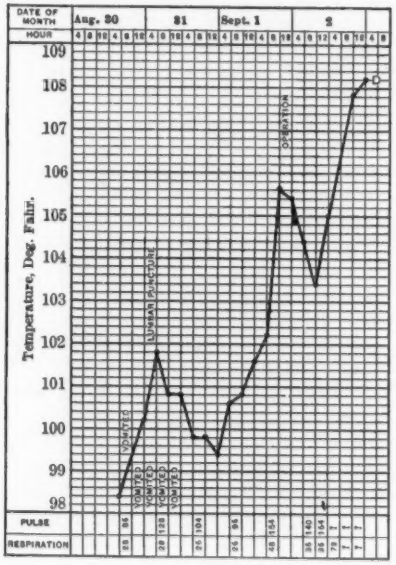

FIG. 11

Cases of operative infection are, of course, not included in this discussion.

6. Fever after Lumbar Puncture:

In one of the patients fever occurred after lumbar puncture. A child sustained a fracture in the parietal region. On admission the patient was drowsy but otherwise exhibited no definite symptoms of any focal lesion. Twenty-four hours later there were indefinite signs referred to the lower extremities: the left seemed to have impaired power and the right was somewhat spastic. Lumbar puncture was done at this point and some bloody fluid was released from the spinal canal. Up to this time the temperature had been normal, but immediately thereafter it began to rise and in an additional twenty-four hours it reached 101.4° F. Operation, which was done at this time, demonstrated extradural bleeding over an intact dura, and was followed, shortly, by a fatality (Fig. 12).

COMMENT. Fever associated with injury of the skull and of the intracranial contents occurs much more commonly than a perusal of the literature would lead one to believe; and while in the presence of some definite infection or other lesion the origin of the temperature is perfectly apparent, in others the cause for the fever is obscure and the mechanism for its appearance, especially when hyperpyrexia is present, is ordinarily said to involve disturbances of the heat-regulating centers.

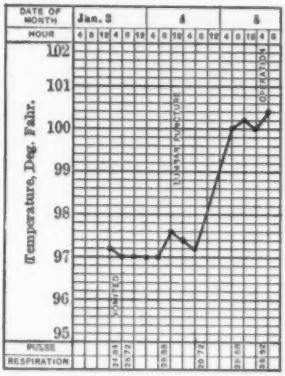

FIG. 12

It was most instructive to compare with these cranial and intracranial injuries a series of cases in which a fracture of the skull could be ruled out but in which the character of the symptoms and the physical findings indicated that a state of concussion (brain contusion) was present. It was cause for much surprise to learn that between 20 and 25 per cent. of the latter cases developed fever of one kind or another during their stay in the hospital. As a general rule the fever was of a very moderate kind and compared with the variety described in group 1. In a very small minority the temperature rose to greater heights. The following cases form excellent illustrations.

Case No. 181734. A young child was thrown in an automobile accident and was brought immediately to the hospital. Except for a small hematoma in the scalp nothing abnormal could be detected and within twenty-four hours the child was perfectly normal, with the exception of the low grade of fever noted in Fig. 13. No adequate cause for the temperature besides the trauma to the head could be discovered. The diagnosis was "concussion."

Case No. 186469. A man, aged forty-three years, was struck in the head by a moving car forty-eight hours before admission to the hospital. He has been in a very dazed condition since, has

complained of much headache and has vomited a number of times. A complete examination in the hospital showed a normal neurological status. With rest in bed the symptoms rapidly disappeared and forty-eight hours later when the man left the hospital no abnormality of any kind could be distinguished. There was absolutely no evidence of any skull fracture.

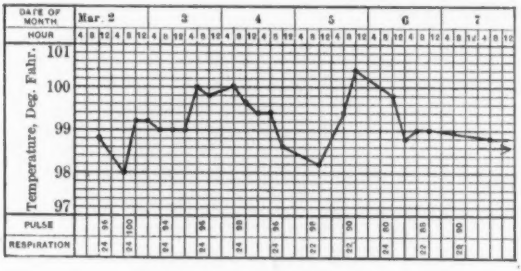

FIG. 13

The fever which this man developed in the hospital is extraordinary. Immediately on admission the temperature (Fig. 14) was 102° F.; within a few hours it rose to 104° F., and thereafter it gradually fell until the time of discharge from the hospital. No other cause beside the head trauma could be demonstrated which could adequately account for the fever. The final diagnosis was "concussion."

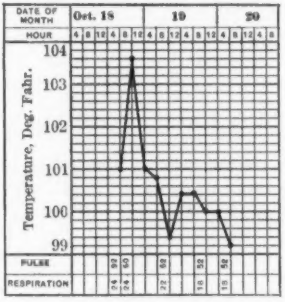

FIG. 14

When fever appears in the cases of this kind, as well as in those in which the brain contusion is associated with fracture of the skull, the assumption seems justified that the more or less widespread disorganization of brain tissue has, probably, primarily involved the heat-regulating centers, and that any fever which appears is due to

the latter disturbance. Implication of these centers could very possibly result secondarily from a general intracranial compression resulting from a sudden cerebral edema following the trauma or more slowly when the compression is due to other causes. Fever under these circumstances would be liable to be a hyperpyrexia. On the other hand there are cases with a more moderate or low grade of fever in which the symptoms and physical findings indicate a more restricted disorganization of brain tissue; possibly in these the temperature finds its origin in the inflammatory process (encephalitis?) essential to the proper repair of the injury.

It is quite possible that in some of the patients with fracture of the skull the phenomenon of fever is connected with the absorption of extravasated blood. In fractures of the long bones an extensive hematoma formation not infrequently is accompanied with a low grade of fever which persists for a short time.

In a series of 61 uncomplicated and closed fractures of the long bones fever was present in approximately one-third. The fever was of a very low grade, usually varied between 100° and 101° F., very rarely reached above 101° F., and never presented the bizarre forms described above with the cases of fracture of the skull or concussion of the brain.

Confirmation of this observation is found in the clinical courses of hematomata of the buttocks: quite commonly these are associated with an intermittent fever which is liable to continue to mount to 103° and 104° F. daily in the absence of any demonstrable infectious or suppurative process until the clot is totally absorbed. A drop of the temperature frequently follows the aspiration of the liquefied portions of the clot, and it is often necessary to incise freely into the hematoma before the temperature disappears; evidently the fluid carries with it some factor which serves to keep up the fever. It seems quite reasonable to assume that a similar mechanism is functionating in some of the cases of fever following a head injury, and the assumption seems especially applicable to the cases of basilar fracture, with hemorrhage into the middle and posterior fossæ of the skull. In these patients lumbar puncture almost invariably releases a bloody fluid which is bacteriologically negative, and which, microscopically, shows very few cellular elements outside of the red blood cells; and the indicated findings serve to negative any impression of an infectious meningitis which might seem probable because of the exhibition of more or less marked symptoms of meningeal irritation. As anticipated in the notes of Case No. 193199—which seems to be an excellent example of this combination of circumstances—the probabilities are very strong that the fever owes its origin to the absorption of extravasated blood, and the physical findings indicate irritation of the meninges by the presence of a foreign fluid.

Our attention to the question of fever after cranial and intra-

cranial injuries was stimulated by some work of Weed and his co-workers. Briefly stated their work consisted of the production of an artificial bacteremia, after which cerebrospinal fluid was released by lumbar puncture; the procedure was followed by a cerebrospinal meningitis. Confirmation of this experimental work in clinical medicine was recently reported by Wegeforth and Latham, who found in a number of general and pulmonary pneumococcus infections that the release of normal cerebrospinal fluid by lumbar puncture was followed by meningeal infection; they describe five cases of their own and refer to others in the literature. Our Case No. 188349 seems to belong somewhere in this group; in this patient a lumbar puncture, with the release of some bloody fluid, practised approximately twenty-four hours after a cranial and intracranial injury, was followed by a rise of temperature. Apparently this seems to be a mild case of infection which, perhaps, under another subsequent course of events might have spontaneously disappeared. The source from which the infection took its origin could very possibly be a dormant one and lie hidden in the tonsils, the teeth or other similar locality. The absence of any true purulent meningitis would seem to indicate that mild cases of infection can occur from which retrogression can be spontaneous. Since the appearance of the communication of Weed we have been very careful and very backward about doing lumbar puncture after cranial and intracranial injury.

In considering the mechanism of fracture of the skull, with intracranial injury, attention was attracted to the fact that a release of cerebrospinal fluid must also occur into the adjacent tissues when a communication is established by dural laceration with the subarachnoid space. An analogous situation is established to the release of cerebrospinal fluid by lumbar puncture; and it is not unreasonable to assume, further, that in the presence of some hidden or dormant focus of infection in the body, either nearby or at a distance, fever would follow. The assumption offers a very satisfactory explanation for the initial rises of temperature which are commonly found after fractures of the skull; it offers an equally plausible explanation for the sudden and sharp rises of temperature which are sometimes seen to follow decompression or other cranial operations in which the dura is opened and cerebrospinal fluid is released from the subarachnoid space. The fact that the process does not terminate always in a suppurative stage should not appear extraordinary, and the retrogression is probably due to the fact that the organism is possibly accustomed to the variety of infection which is lighted up or that the body's natural resistance powers are amply sufficient to quickly bring the process under control. In any case the insult of lumbar puncture, trauma or operative interference, with a subsequent release of cerebrospinal fluid, seems to create a *locus minoris resistentiæ* and the injured area forms a

fixation-point for any pathogenic organisms which, at the moment, are circulating in the blood-stream.

It seems very probable, when all things are considered, that in any particular case more than one of the factors indicated in this communication may be operative to account for any fever which develops. Only in a comparatively few cases is it possible for one to put one's finger accurately upon the actual mechanism.

SUBCUTANEOUS EMPHYSEMA: A COMPLICATION OF INFLUENZA PNEUMONIA. REPORT OF SEVEN CASES.

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Recent clinical reports have mentioned subcutaneous emphysema as a rare complication of influenzal bronchopneumonia. Thus, Symmers[1] records 3 cases; Berkley and Coffen[2] 11; Clark and Synnott[3] 12; Blanton and Irons[4] and Stone and Swift[5] merely mention its occurrence. Leichtenstern,[6] in his exhaustive monograph on influenza, makes no note of this complication. Our own experience comprises a total of 9 cases, of which we have studied 7 cases carefully, and these are herewith presented. In about 3000 cases of influenzal pneumonia subcutaneous emphysema occurred in 0.3 per cent., and in the entire series of over 12,000 cases of influenza in only 0.07 per cent.

Clinical Features of Influenza Pneumonia. The clinical picture of influenza pneumonia has been so often described that we do not deem it necessary to repeat it here. Our experiences, on the whole, correspond to all previous reports. We desire to emphasize, however, certain features of the disease, not only because they were apparently characteristic of the picture but because they may be significant factors in the production of emphysema.

1. *Toxemia.* This was striking early in the epidemic and entirely out of proportion to all physical findings.

[1] Jour. Am. Med. Assn., 1918, lxxi, 1482.
[2] Ibid., 1919, lxxii, 535.
[3] Am. Jour. Med. Sc., 1919, clvii, 219.
[4] Jour. Am. Med. Assn., 1918, lxxi, 1988.
[5] Ibid., 1919, lxxii, 487.
[6] Influenza, 1912. Alfred Hölder, Wien u. Leipzig.

2. *The Paroxysmal Cough.* This occurred early, was particularly distressing and closely resembled the cough of pertussis, excepting the absence of the "whoop" at the end of the paroxysms.

3. *Epistaxis and General Tendency to Hemorrhage.*

4. *The Frequency of Pain in the Chest.* The rapidity with which this developed was particularly noticeable, physical examination failing to reveal any cause.

5. *Cyanosis and Dyspnea.* The purple hue of the face, upper chest, and finger nails, at once suggested cardiac failure, but on physical examination, heart tones were generally good and there was usually no evidence of right heart failure. Blood-pressures were practically normal.

6. *The Relative Paucity of Physical Findings and the Rapidity with which the Signs of Consolidation Developed.* It is noteworthy that on admission a few crepitant rales were heard posteriorly. Percussion revealed a distinctly hyperresonant note, and this had no apparent relation to the amount of consolidation.

REPORT OF CASES.

CASE I.—Lieut. P. Taken ill and admitted October 2, 1919, with headache, sore-throat and general aching. Physical examination revealed a young, fairly well-nourished man, face congested, pulse of fair quality, temperature 103°, respirations 24. Many crepitant rales in right lower lobe; impaired resonance and diminished breath sounds.

October 4. Dulness over the right lower lobe; bronchovesicular breathing. The roentgen-ray report showed spot shadows in the lower right.

October 9. Slight cyanosis of nose, lips and ears and the physical signs of consolidation in right lower lobe. On the left side many crepitant rales. At 2 P.M. examination of the chest revealed loud crackling sounds over the apices. On palpation of these areas a fine crepitation was obtained and a diagnosis of subcutaneous emphysema made. The extent of the involvement at this time was the left supraclavicular space and upper part of the left chest. The patient had no complaint of precordial or pleuritic pain at this time. On repeated questioning he could not associate his swelling in the neck with any cough; the patient was unaware of it until it was mentioned to him by the physician. The temperature showed no alteration from that on the previous day. The respirations were not changed in rate or excursion. The roentgen-ray report on this date revealed a shadow in the right lower lobe; no evidence of pneumothorax.

October 10. The air extended bilaterally and involved the chest, neck and upper part of the abdominal wall, interfering with examination of the chest.

October 13. The emphysema now involved the left side of the face. The left eye was closed because of emphysema of the lids.

FIG. 1.—Note emphysema of left eyelid.

FIG. 2.—Note emphysema of supraclavicular tissues.

(Fig. 1). The upper and lower extremities were also involved. The scrotum was distended to about one and one-half times its normal size. The patient had the appearance of a nephritic.

Roentgen-ray Reports. October 14. Extensive quantities of "gas" shown in tissues of chest, neck (Fig. 2), arms down to wrist,

FIG. 3.—Note emphysema of neck and upper chest.

FIG. 4.—Note emphysema of neck.

over abdominal muscles and through the pelvis, scrotum and legs (Fig. 6).

October 17. Spot shadows throughout both lungs. Dilated heart. "Gas" in quantities throughout trunk, pelvis, legs and arms and wrists.

October 19. "Gas" in the legs has diminished; chest and abdomen remained the same.

FIG. 5.—Acute ulcerative bronchiectasis; note the apparent necrotic condition of the walls.

October 14. Pneumococcus Type I reported in the blood and patient was given 100 c.c. antipneumococcus serum; this was repeated on the next day.

October 16. Patient complained of pain in the right side.

October 17. Fifty cubic centimeters of antipneumococcus serum. No difficulty in locating veins for intravenous injection and no apparent increase in the emphysema after the severe chill following the initial injection of serum. Patient showed no improvement in

general condition, but emphysema diminished, particularly about the face.

October 20. Patient died.

Blood culture: Pneumococcus Type I. Leukocytes: October 3, 4000; October 10, 3400; October 15, 10,000. Anaërobic and aërobic cultures of "gas."

The skin was punctured under aseptic precaution and sterile salt solution introduced by capillary pipette. The washings were then cultured. Negative results after seven days' incubation at 37° C.

FIG. 6.—Ulcerative bronchitis; infiltration with air of peribronchial tissues. Note the torn, necrotic condition of the bronchial wall and the distention of the peribronchial areolar tissues.

Necropsy (18–196). Subcutaneous emphysema; left eyelids, left temporal region, cheeks and entire neck, entire chest and abdomen to anterior scapular line without involving gluteal region, left side of scrotum, left lower extremity to knee, right lower extremity to thigh. Subfascial and fascial emphysema of neck, axilla, chest and abdomen. Slight icterus. Emphysema of mediastinum. Edema of areolar tissue of mediastinum. Acute fibrinous pleuritis, right. Early lobular bronchopneumonia in left upper lobe. Confluent bronchopneumonia in left lower lobe. Compensatory inflation on anterior edge of entire lung, left. Bronchopneumonia, upper and lower lobe; compensatory inflation, anterior edge, right lung. Purulent bronchitis with acute ulcerative bronchiectasis, more marked in left lung. Acute fibrinopurulent bronchitis. Cloudy

swelling and slight right-sided dilatation of heart. Acute follicular splenitis. Congestion and edema of adrenals. Acute parenchymatous nephritis. Cloudy swelling and terminal congestion of liver. Cloudy swelling and congestion of the pancreas. Lymphoid hyperplasia and congestion of intestines. Edema of pia-arachnoid; congestion of brain.

Microscopic Findings: Lung: fibrinopurulent bronchopneumonia; areas of hemorrhage; marked edema; acute ulcerative bronchiectasis; dilatation of peribronchial spaces; areas of necrosis; acute emphysema. Heart: congestion; cloudy swelling; conglutination and hyaline thrombi; acute fibrinous pericarditis. Spleen: acute diffuse splenitis; massive hemorrhages; partial necrosis of reticulum. Adrenal: lipoid exhaustion; congestion. Kidneys: acute glomerulotubular nephritis. Liver: congestion; cloudy swelling; focal toxic necrosis. Brain: cloudy swelling of ganglion cells; congestion.

Case II.—Private M. Admitted February 18, 1919. Previous history: measles, pertussis, scarlet fever. Onset: two days before admission he complained of cough, chilly sensation, fever. On admission: a young, well-nourished man, apparently not very sick, complaining of headache, pain in the extremities and cough. Examination: no definite lung finding. A diagnosis of influenza was then made on the basis of general appearance. Patient showed no change until three days later, when many rales were noted throughout the chest.

February 22. Left lower dulness, distant bronchial breathing and fine crepitant rales; right lower dulness and rales.

February 24. Complained of intense pain in the right chest. Examination showed dulness, bronchial breathing, rales; no evidence of friction rub. On the left side the physical findings were unchanged, but notation was made of diminished breath sounds on left side.

February 25. Condition poor, pulse could not be obtained, cyanosis intense; hyperresonance over left chest anteriorly; in the left midaxillary line: dulness, breath sounds present. The heart borders could not be outlined on percusion; no visible apex impulse. On auscultation over the pulmonic area a loud crackling sound was heard as the stethoscope was placed on the skin. On palpating this area was distinct; fine crepitations in the subcutaneous tissue were obtained. This area extended from the second intercostal space to the nipple proper. The breath sounds were diminished; the heart tones were not audible. The clinical picture at once suggested a pneumothorax with subcutaneous emphysema. 4 P.M.: The emphysema had spread to the clavicle on the left side and could be felt over the sternum. It also extended to a slight degree below the left nipple at 6 P.M.: subcutaneous emphysema had extended laterally on the left side and reached the midaxillary line. The

patient was very cyanotic. The upper extremities were cold and covered with a clammy perspiration. Patient complained of pain and numbness of feet; this complaint commenced at 12 P.M. The feet were cold; there was no pulsation in the dorsalis pedic artery, but the femoral pulse and sounds could be obtained. The abdomen was tender and slightly distended. 7 P.M.: emphysema had not spread; pulse could not be obtained. 8. P.M.: patient died.

Roentgen-ray Reports. 1. Spot shadow, right lower, February 18. 2. "Gas" in the subcutaneous tissue of left chest. No evidence of pneumothorax.

Laboratory Reports. Sputum: Streptococcus hemolyticus. Blood Culture: Negative. Leukocytes: February 24, 6900.

Necropsy (19–364). Subcutaneous emphysema of neck, left upper chest, upper part of left abdomen. Emphysema of anterior mediastinum. Left pneumothorax. Zenker's hyaline degeneration of abdominal muscles. Bilateral acute fibrinous pleuritis. Confluent bronchopneumonia of left basal lobe. Patchy bronchopneumonia of left upper lobe. Inflation of lung borders. Confluent bronchopneumonia of right upper and lower lobes; inflation of borders. Purulent bronchitis. Cloudy swelling and dilatation of right heart. Acute follicular splenitis. Congestion of adrenals. Congestion of kidneys; acute parenchymatous nephritis. Acute tonsillitis. Cloudy swelling of liver. Cloudy swelling of pancreas. Slight congestion of intestines. Slight congestion and edema of pia-arachnoid. Petechial hemorrhages of brain.

Microscopic Examination. Lung: fibrinocatarrhal bronchopneumonia. Ulcerative bronchitis. Marked congestion. Heart: slight congestion; cloudy swelling. Spleen: acute follicular splenitis. Adrenal: lipoid exhaustion; edema; congestion. Kidneys; extreme congestion; acute tubular nephritis. Liver: cloudy swelling; biliary pigmentation. Brain: focal hemorrhage; cloudy swelling of ganglion cells.

CASE III.—Private B. Admitted October 3, complaining of sore-throat.

October 9. Many rales left base posteriorly; temperature, 104.4; pulse, 112; respiration, 48.

October 13. Patient was reported as a case of subcutaneous emphysema and notations on the brief showed it had developed on the left side, upper part of the chest, on the evening of October 13.

October 14. Examination showed a very robust young man, cyanotic and dyspneic, very uncomfortable and toxic. The emphysema involved the face on both sides and the patient had the appearance of a case of mumps; palpation revealed definite crepitation. The eyelids were not involved except the right, which was but slightly swollen. Both sides of the neck were distended (Fig. 3). "Gas" was also present in the subcutaneous tissue of the chest anteriorly and posteriorly. The abdomen, anteriorly, appeared

distended; the skin was "ballooned" up from the underlying tissues as if a layer of air were interposed; on deep palpation crepitation was obtained. Both upper and lower extremities were involved; the scrotum was distended. There was no change in the clinical picture until October 17, when the patient began to complain of pain in the region of the larynx. Laryngoscopic examination on October 18 showed congestion of larynx, with some purulent secretion; cords normal.

October 19 to 24. The emphysema gradually diminished and the patient appeared to be improving.

October 25. Complained of pain on swallowing and dyspnea.

October 26. Exploratory aspiration of the right chest; needle used for local anesthesia was used as aspirator, no fluid but air returned. A larger needle was introduced and returned a large amount of air without blood or fluid. Comfortable during and immediately after aspiration. Thirty minutes later intense dyspnea, rapid pulse, dilated pupil.

October 27 and 28. Emphysema diminishing, condition poor; influenza immune serum intravenously.

October 29. Patient died.

Roentgen-ray Reports. October 14. Roentgen ray; extensive quantities of air throughout subcutaneous tissues of neck and chest, apparently following fascial planes over the abdomen, pelvis and gluteal region, right arm to elbow and left arm to elbow, left leg below knee and right leg below knee. Extensive spot shadow throughout lungs.

October 16. Gas showed more extensively throughout body (Fig. 4), more particularly in arms and legs.

October 17. Spot shadows throughout both lungs. Dilated left heart. Gas over the trunk, and arms to wrist, and legs half-way below knee.

October 18. The gas throughout tissues over trunk somewhat less prominent, but well marked in right arm.

October 19. Extension of gas in right hand. Chest and abdominal condition improved.

October 21. Gas in tissues over chest, neck, abdomen, thighs and arms appeared to be less than on previous day.

Laboratory Reports. White counts: October 16, 16,200; October 18, 17,500; October 25, 13,900. Sputum: Non-hemolytic streptococcus; staphylococcus; pneumococcus (type not determined). Urine: Many hyaline and granular casts. Specific gravity, 1028.

October 14. All cultures, aërobic and anaërobic, negative.

Necropsy (18–216). Subcutaneous emphysema of entire chest and abdomen. Prelaryngeal abscess. Acute bilateral fibrinous pleuritis. Confluent bronchopneumonia of both lungs. Compensatory inflation of anterior edges of both lungs. Acute purulent bronchitis and bronchiectasis. Acute diffuse splenitis. Cloudy

swelling and congestion of kidneys. Cloudy swelling and terminal congestion of liver. Acute adenitis of mesenteric lymph nodes. Acute myositis and hyaline degeneration in rectus muscles. Slight edema of pia-arachnoid.

Microscopic Examination. Lungs: Fibrinocatarrhal bronchopneumonia; ulcerative bronchitis; areas of hemorrhages; acute emphysema. Heart: congestion and cloudy swelling. Peribronchial lymph nodes: marked congestion; edema; sinus catarrh. Adrenal: lipoid exhaustion; congestion and edema; hyaline thrombosis. Kidneys: congestion; cloudy swelling. Liver: hydrops; congestion; hyaline thrombosis; slight biliary pigmentation.

CASE IV.—Private P. Admitted October 10, 1918. Previous history negative. Was sick three or four days before admission to hospital, complaining of severe backache, pain in the chest, vomiting, slight coryza; throat injected, a few scattered rales in both bases. The patient showed no clinical change until ten days later, when he developed a rigid neck and positive Kernig's sign. A spinal puncture was performed but proved to be negative, microscopically and bacteriologically.

October 21. Patient had marked difficulty in breathing and swallowing; examination revealed an acute laryngitis, acute inflammatory edema of the glottis, epiglottic folds and arytenoids. Aryepiglottic folds and arytenoids were markedly congested, interfering with respiration. High tracheotomy was performed under local anesthesia.

October 24. One of us was called in to see the case at this time and distinct crepitation was present in the tissue of supraclavicular spaces and neck. On observation during the day it was noted that the gas remained localized in neck region but increased in quantity.

October 25. Patient died.

Roentgen-ray Reports. October 12. Spot shadows in both lower lobes. Throat culture: Staphylococcus predominating. Leukocytes: October 12, 5800. Sputum: Staphylococcus; pneumococcus.

Necropsy (18–209). Subcutaneous emphysema of anterior neck. Operative wound of neck (tracheotomy). Cervical abscess. Bilateral fibrinopurulent pleuritis. Bilateral lobular bronchopneumonia of all lobes. Acute diffuse splenitis. Massive hemorrhage of right adrenal. Acute parenchymatous nephritis; congestion of kidney. Cloudy swelling of liver. Congestion and edema of pia-arachnoid. Lymphoid hyperplasia of intestines. Acute suppurative otitis media, right. Acute mastoiditis.

Microscopic Examination. Lungs: acute fibrinocatarrhal bronchopneumonia; edema; marked congestion; ulcerative bronchitis; acute inflation. Heart: congestion; cloudy swelling; peribronchial lymph nodes: congestion and edema; sinus catarrh. Spleen: acute diffuse splenitis. Adrenal: lipoid exhaustion; congestion. Liver: congestion; cloudy swelling. Kidneys: acute glomerulotubular

nephritis. Brain: cloudy swelling of ganglion cells; congestion; minute hemorrhages.

CASE V.—Lieut. S. Admitted to Base Hospital October 3, 1918. Previous history negative.

October 1. Onset, with general malaise and chilliness. On admission a robust young man, rather dyspneic, coughed persistently. Intense cyanosis of face; dulness of right lower lobe; bronchial breathing.

October 7. Extensive involvement of right lower and middle lobes, with dulness, bronchial breathing, crepitant and subcrepitant rales. Many small areas of dulness with many rales in the left side. Cyanosis and dyspnea very pronounced.

October 10. Distant crepitation over the entire right side of the chest and neck. The face was not involved.

October 11. Patient died.

Roentgen-ray Reports. October 8, 1918: shadows involving both lower lobes. Sputum bacteriology: streptococcus, pneumococcus, staphylococcus, M. catarrhalis.

Leukocytes: October 8, 1918, 6500.

CASE VI.—Private G. Admitted October 3, 1918. Previous history: exanthemata, including smallpox, when a child; fracture of ribs several years ago. Onset sudden, cough, pain in chest and fever. On admission: a robust young man, appearing not very sick, with cyanosis of face; throat purple and injected; no definite evidence of consolidation, many rales. Diagnosticated severe influenza.

October 5. Consolidation lower left.

October 8. Subcutaneous emphysema in uper part of chest, neck, face. Patient delirious; petechial eruption on skin.

October 9 to 12. No change; clear mentally.

October 18. Emphysema of subcutaneous tissue diminished; absent on chest.

October 21. Lungs clear; breath sounds high pitched; no rales.

November 1. Discharged from hospital as recovered.

Roentgen-ray Reports. October 14. Extensive quantities of gas in the tissues over neck, shoulders, abdominal muscles. Spot shadows throughout right lung. Shadow in lower left. Gas over gluteal region, thigh, legs and forearms.

October 16. Gas more extensive than on previous report.

October 18. Gas less marked in tissue than on previous report. Leukocytes: 7500.

CASE VII.—Private R. Admitted October 3, 1918, with diagnosis of influenza.

October 9. Many rales over right lower lung.

October 14. Developed bilateral lobular pneumonia.

October 15. Patient very weak, coughed persistently, sputum bloody.

October 16. Cyanosis of face. Mental condition sluggish.

October 18. Patient appeared very toxic and was delirious. Spinal puncture performed and 20 c.c. of clear fluid removed; this showed no increase in cells and no bacteria; globulin negative.

October 20. Condition serious; patient dyspneic and cyanotic. Subcutaneous emphysema developed, involving the face, neck, chest and upper and lower extremities bilaterally.

October 21. Patient's condition serious. The emphysema in the lower extremities diminished.

October 22. Subcutaneous emphysema had extended to waist line. Lower extremities free.

October 23. Subcutaneous emphysema increasing.

October 25. Intravenous injection of immune serum.

October 26. Weak; complains of pain in chest.

October 29. Emphysema disappearing. Gradual improvement. Discharged from hospital November 22, 1918, as cured.

Roentgen-ray Reports. October 15. Shadow in lower right.

October 20. Extensive spot shadows in lower right with same on left. Gas showing throughout muscles of trunk with some in the thighs, also in forearms.

October 21. Gas in tissues of neck, chest, abdomen and arms.

October 23. Gas extending.

In addition to the above one of us has observed a similar condition complicating measles. The emphysema was localized in the chest wall; the patient recovered. In another instance generalized subcutaneous emphysema developed postoperatively in a patient who had a pyopneumothorax. In this case the emphysema developed one day after operation; the patient was aspirated repeatedly prior to operation but never presented evidence of subcutaneous emphysema. When the emphysema did develop it was rapid and generalized, involving face, neck, chest, abdomen and extremities. This patient died.

Clinical Analysis. An analysis of these cases from the clinical aspect reveals certain constant features.

The onset of the subcutaneous emphysema was apparently sudden, but after its appearance the "gas" spread relatively slowly, attaining its greatest extent in twenty-four to forty-eight hours.

The time of appearance of the emphysema, relative to the stage of influenza pneumonia, was not definite, but it is safe to say that in the majority of the cases the "gas" developed when the pneumonic process was well advanced. Table I shows this relationship.

TABLE I.—TIME OF DEVELOPMENT OF EMPHYSEMA.

Case No.	Influenza.	Pneumonia.	Emphysema.	Death or recovery.
I . .	October 2	October 9	October 9	October 19
II . .	February 18	February 22	February 25	February 25
III . .	October 3	October 5 or 9	October 14	October 29
IV . .	October 10	October 10	October 24	October 25
V . .	October 3	October 5 or 7	October 10	October 11
VI . .	October 3	October 5	October 8	Recovered.
VII . .	October 3	October 14	October 20	Recovered.

It is noteworthy that the paroxsymal cough, to which has been ascribed an important place in the possible etiology of the subcutaneous emphysema, occurs very early in the picture of influenza pneumonia, and that when the pneumonic process is well established cough is not common. It might be assumed that with a generalized pulmonary emphysema, or with a destructive softening of the lung parenchyma (Torrey and Grosh[7]) and an associated severe cough, rupture of alveoli could easily occur and be the source of emphysema. The striking contrast, however, of the late appearance of the emphysema and the absence of cough during that period suggest that the cough probably plays a minor role.

Symptoms associated with the development of the subcutaneous emphysema were, according to our experience, practically negative. The patient was generally unaware of the complication until it was pointed out to him. The temperature, pulse and respiration showed no change. Torrey and Grosh,[7] however, state that "when in apparently respiratory extremis a patient would complain of pains, substernal and in the jugular fossæ, and crepitation would be noted in the subcutaneous tissues at the root of the neck, an immediate marked subjective relief was apparent, rapidly followed by a noticeable improvement in the respiratory excursion of the chest and the most striking decrease in cyanosis and jugular distention." Measurements of the respiratory excursion showing "that there was apparent an increase in the chest circumference during the illness and an abnormally small respiratory excursion, with a striking increase immediately as the subcutaneous emphysema gave vent to the air contained in the mediastinum."

We observed precordial pain in but one case, but pleuritic pain was common. Cyanosis was not relieved to any marked degree nor was the respiratory rate diminished as shown by our clinical record. Table II shows that the temperature, pulse and respiratory rate, before and after the appearance of the subcutaneous emphysema, were practically unchanged.

Location of Subcutaneous Emphysema. The subcutaneous emphysema has developed most frequently in the neck region. In one case it was first noticed in the region of the second left intercostal space. The supraclavicular spaces, neck, chest, face, extremities, abdomen and scrotum, have been involved. It is easily diagnosticated by obtaining a fine crepitation on palpation, and on auscultation the stethoscope produces a loud crackling sound as it comes in contact with the skin.

Absence of Signs of Inflammation. There is no apparent change in the leukocytes with the development of the emphysema. Likewise there is no local skin eruption or inflammation. The physical condition seems but slightly affected by the development

[7] Am. Jour. Med. Sc., 1919, clvii, 170.

TABLE II.—TEMPERATURE, PULSE, AND RESPIRATION BEFORE AND AFTER APPEARANCE OF EMPHYSEMA.

Date.	Before emphysema developed.			Date.	After emphysema developed.		
	Temp.	Pulse.	Resp.		Temp.	Pulse.	Resp.
CASE I:							
Oct. 7	105.0	112	28	Oct. 9	103.0	110	26
	100.0	92	26		101.0	90	30
Oct. 8	104.0	116	24	Oct. 10	101.8	84	24
	101.4	100	26		101.8	100	26
				Oct. 15	101.8	110	30
					100.2	84	40
Feb. 22	100.0	92	22				
	102.2	92	22				
CASE II:							
Feb. 23	102.8	90	24				
	102.0	110	28				
	102.6	108	24				
	102.6	92	24				
	103.0	112	26				
Feb. 24	103.6	90	24	Feb. 25	103.2	120?	36
	104.0	108	46		104.0	120?	36
	104.4	136	42		100.0	?	40
	103.8	120	36		102.0	?	48
	102.0	120	36				
CASE III:							
Oct. 10	104.4	102	48	Oct. 13	100.0	116	44
Oct. 11	103.6	104	48		101.0	118	40
	103.8	112	36	Oct. 14	98.0	70	26
Oct. 12	101.0	98	36		100.0	110	30
	101.6	140	40	Oct. 15	100.0	100	22
					100.0	100	22
				Oct. 16	101.0	120	28
				Oct. 17	98.0	118	36
					101.0	120	30
CASE V.							
Oct. 8	103.0	98	36	Oct. 10	103.0	122	54
	102.0	140	38		102.0	120	56
	104.0	120	28				
Oct. 9	103.6	120	46				
	103.0	140	60				
CASE VI:							
Oct. 7	101.0	126	30	Oct. 8	101.0	96	30
	102.0	100	46		102.0	120	32
				Oct. 9	100.0	98	42
					102.6	120	42
				Oct. 10	101.2	96	30
CASE VII:							
Oct. 18	99.2	106	36	Oct. 20	103.2	144	48
	99.8	100	32		102.2	120	32
	101.2	110	44	Oct. 21	100.4	130	40
Oct. 19	99.0	120	46		102.4	128	52
	99.4	120	52		102.2	120	54
	98.8	140	52		102.8	120	44
	100.0	126	40	Oct. 22	101.0	104	44
					101.0	124	56
					101.2	112	50
					101.2	122	54

of even most extensive emphysema. In all of the patients the emphysema subsided considerably as the disease progressed and death was always due to associated conditions. Two of the cases recovered entirely.

Of the cases which developed subcutaneous emphysema it is noteworthy that in addition to the severity of the picture of pneumonia there were additional complications. Thus delirium occurred in one patient, meningismus in another and possibly a slight pneumothorax in still another. Prelaryngeal abscess and edema of the glottis were also encountered. The association of subcutaneous emphysema following tracheotomy has been reported previously,[8] but not in association with pneumonia. In Case IV the occurrence of an emphysema following tracheotomy is noteworthy, since wounds of the larynx and trachea[9] have been given as a cause of emphysema, but it seems evident that the emphysema in Case IV was not due to the laryngeal pathology.

In attempting to elicit a possible etiological factor in these cases we questioned our patients as to trauma and previous history of gas attacks. In none of these was any such history obtained.

The occurrence of only one case of pneumothorax diagnosticated clinically and the constant absence of any evidence of pneumothorax on roentgen-ray examination in all our cases of subcutaneous emphysema is emphasized.

TREATMENT. The treatment in general was directed to the pneumonia or the associated complication rather than to the emphysema. As has been stated the development of the subcutaneous emphysema was not accompanied by any symptoms. The emphysema apparently will diminish or disappear without any operative or medicinal interference.

DISCUSSION OF ANATOMICAL FINDINGS. The subcutaneous emphysema in two of the cases was considerably more marked on the left than on the right side. The significance of this will be commented on later. The external appearance of the bodies has been described in the clinical part of this paper, and it need only be added that two of the patients presented the extreme lividity of the face and the body so frequently seen in fatal influenza.

The subcutaneous tissues when incised were moderately moist and slightly bloody, and, in a general way, had the appearance of innumerable small air bubbles in the alveolar tissue. Over the chest this distended areolar tissue measured about 15 mm. in its thickest part, over the abdomen about 5 mm. Sections from this tissue presented none of the changes associated with inflammation. Coagulation necrosis of the rectus muscles was met with in two cases;

[8] Ashley and Wright: Diseases of Children, American edition, 1893. Quoted by Alexander, M. E., and Follett, E. C.: Jour. Am. Med. Assn., 1919, lxxii, 930.

[9] Lexer, E.: General Surgery. Quoted by Alexander, M. E., and Follett, E. C.: Jour. Am. Med. Assn., 1919, lxxii, 930.

this condition was also present in non-emphysematous influenzal patients.

The mediastinum in two of the bodies resembled the subcutaneous tissues in general appearance. In two other bodies no changes were noted. In the first patient the areolar tissue throughout the mediastinum was edematous as well as emphysematous, forming a large mass over the pericardium, which, when dissected out, was found to weigh 165 grams. Otherwise the gross and minute anatomical changes in the various organs were similar to those in other influenza cases, and since a study of this disease has been published by one of us,[10] it would lead to unnecessary repetition to enumerate them here. We shall confine ourselves, therefore, to a description of the respiratory tract lesions, and these, for the sake of brevity, are discussed collectively.

Lungs. It may be stated that the lungs differed in no way from the lungs of other influenzal patients. The left lungs varied from 490 to 1040 grams in weight and the right lungs from 590 to 850 grams. The anterior edges of the lungs were in each case moderately inflated, but this was never more marked than in other patients which did not have subcutaneous emphysema. In no case were air bladders or pearl-like distended air vesicles seen, although this was not infrequently encountered in our other influenza autopsies. The surface of the lungs was carefully examined to note any point of possible rupture, but this could in no instance be demonstrated. The posterior parts of the lungs were more or less consolidated, the areas of bronchopneumonia tending to become confluent. The extent of pneumonic involvement was never more marked than in the average influenza pneumonitis. The pleuritic complications were of the type usually met with. In no instance were the lungs firmly adherent to the lateral chest wall.

The cut surface resembled that usually seen in influenza pneumonitis, that is, it was generally very bloody and very moist, with large, poorly circumscribed, consolidated areas, some of which were granular, others entirely smooth. The bronchial branches, in all but one instance, contained large quantities of thick, creamy pus. The bronchial mucosa was swollen and deeply hemorrhagic. Once the walls of several of the larger bronchial branches were definitely eroded and the lumen markedly dilated, forming acute ulcerative bronchiectases (Fig. 5). In another instance cylindrical dilatation of some of the bronchial branches was present.

The microscopic examination of the lung presented the usual hemorrhagic, fibrinocellular exudate of influenzal pneumonitis. In all but one instance there were areas of necrosis, usually surrounding a destroyed bronchiole, but occasionally seemingly independent

[10] Lucke, Baldwin, Wright, Toynbee and Kime, Edwin: Pathological Anatomy and Bacteriology of Influenza, Arch. Int. Med., August 15, 1919.

of the bronchial tubes. Once marked thickening of the interpulmonary septa occurred. The bronchial branches in each instance presented here and there partial or complete erosion of their walls, masses of necrotic debris and fibrin occupying the site of the destroyed areas. Once the peribronchial tissue was found markedly distended, giving the appearance that air had escaped through the destroyed bronchial wall in the loose peribronchial tissue (Fig. 6). While ulcerative bronchitis was not uncommonly met with in about 200 cases of influenzal pneumonitis, which we have examined here postmortem, it appeared regularly in the four cases complicated with subcutaneous emphysema. Ulcerative bronchiectasis of the larger bronchial branches was uncommon in the epidemic here, occurring only twice outside of the case reported here.

Bacteriological cultures were made during life from the subcutaneous tissues at various parts of the body, and after death from the lungs, pleural exudate, subcutaneous tissues, heart's blood, spleen, brain, and other organs. As has been stated in the clinical part of this communication, all the antemortem cultures were negative; the postmortem cultures presented in each case hemolytic streptococci, pneumococci, and influenza bacilli, either alone or in combination.

DISCUSSION OF THE MODE OF ORIGIN OF SUBCUTANEOUS EMPHYSEMA. It may be well to briefly review the various theories which have been suggested as the cause of this unusual complication:

Bacterial activity may be at once dismissed, since no local inflammatory reactions point to this condition. Torrey and Grosh speak of a destructive softening of the lung parenchyma, due probably to alteration of the lung tissue by toxin or virus. Chemical changes in the blood, permitting escape of air into the tissues is a possibility. The delayed time of the coagulation of the blood, the frequency of nose-bleed, petechial hemorrhages throughout the body, allows one to entertain the thought that chemical blood changes may occur which prevent the blood from maintaining the normal balance of its gases. We have analyzed the air obtained by puncture of the distended scrotum of one of the patients examined and found it to resemble atmospheric air.

Most of the writers who have reported cases of subcutaneous emphysema complicating influenza maintain that this condition is purely mechanical in origin. Thus, Symmers says, "In two cases rupture of the distended air vesicles has taken place probably near the apex of the lung. In another rupture occurred in the left pleural cavity, with the production of pneumothorax. In none of these cases was it possible to demonstrate at necropsy the actual point of rupture." Berkley and Coffen report 11 cases, 2 of which were associated with spontaneous pneumothorax. They suggest that subcutaneous emphysema may occur through the intrapleural route, providing that adhesions exist between the two layers of

pleura at the point of rupture, or through the extrapleural route if the air dissects its way to the hilum of the lung. Clark and Synnott, in the report of 12 cases, conclude that the origin of the gas was from weakened alveoli of the lung under pressure.

We believe that subcutaneous emphysema chiefly arises by escape of air from ulcerated or eroded bronchi and its passage along peribronchial or perivascular channels into the mediastinum. The clue of our explanation, in at least 2 of the cases, was found in the occurrence of marked ulcerative bronchitis and subcutaneous emphysema, both located on the left side.

If we disregarded simple mechanical rupture of peripheral alveoli as a highly improbable factor in the production of subcutaneous emphysema, there remain, then, only three possible ways for pulmonary air to reach the subcutaneous tissues: (*a*) by necrotizing destruction of alveoli; (*b*) by ulcerative or erosive rupture of a bronchus; (*c*) by rupture of pulmonary tissue through adherent pleural surfaces. By necrotizing destruction, alveolar air could pass into the loose peribronchial or perivascular areolar tissue and thence into the mediastinum. By ulceration or erosion of a bronchus the air would likewise gain the mediastinum. In neither case would the air pass into the pleura, because this is a closed sac. The third way is through perforation of necrotic pulmonary and overlying pleural tissue into the pleural cavity, resulting in pneumothorax. Should the visceral and parietal layers of the pleura adhere to one another the air could penetrate both layers and dissect the areolar tissue between the parietal pleural and thoracic wall to the mediastinum. Such a case would necessitate, it seems, a considerable area of adhesion. Once the air has found a vent from the pulmonary tissues, expiratory force and elasticity of the chest wall would further drive it along the lines of least resistance, which is into the neck (the location in which the subcutaneous emphysema apparently always began). Thence, along fascial planes, the emphysema would become generalized.

Microscopic examination presented a distention of the peribronchial areolar tissue (Fig. 6), and this we consider a support of our second explanation of the escape of air from the lung.

The question would naturally arise, "Why should this condition be so uncommon when ulcerative bronchiectasis is not infrequently met with in various diseases, as, for instance, tuberculosis. But it must be pointed out that bronchiectasis rarely occurs acutely and that the reaction of the tissues surrounding the bronchi would probably form a wall impenetrable to the air. It may be assumed, then, that only in rapidly occurring bronchiectasis, without sufficient tissue reaction, is such a condition possible, and that they arise but rarely. It would seem, likewise, that a fair-sized bronchial branch is necessary to allow sufficient air to escape for the production of emphysema. This would appear to us more likely as the cause than

mechanical rupture of peripheral alveoli, for not only are hugely distended and sometimes ruptured alveoli frequently encountered in both acute and chronic pulmonary emphysema, but the rupture of a few air sacs would not likely lead to a widespread air infiltration. Further, rupture of peripheral alveoli could only produce emphysema, provided that firm adhesions were present between the pleural layers at the point of rupture. Pneumothorax would result otherwise and in none of the cases reported has this occurred frequently. In the one instance where pneumothorax was present in our series, it was not at all marked, since only a few small air bubbles escaped upon puncturing chest wall through a pocket filled with water.

It is not at all unlikely that erosion or rupture in a bronchial tube might be overlooked, and this, we feel, rather strengthens than weakens our argument. The absence of mediastinal emphysema in two of our cases may be readily explained by the gradual absorption of air which occurred in every one of these cases, for, as has been stated, a marked reduction in the extent of the subcutaneous emphysema took place in every instance. This probably is due to the gradual closing up of the point of eruption because of cellular reaction.

CONCLUSIONS. 1. Subcutaneous emphysema is an exceedingly rare complication of influenza.

2. Previous explanations of the condition seem inadequate and unsatisfactory.

In two of our postmortem cases we encountered a combination of more pronounced left-sided subcutaneous emphysema with left-sided ulcerative bronchitis, and on microscopic examination the peribronchial tissue was found greatly distended. We believe the bronchial air to have escaped into the loose peribronchial tissues, and by dissecting along lines of least resistance to have gained entrance to the mediastinum and thence, along fascial planes, to the subcutaneous tissues. In this way the closed pleural sacs are not involved, and this accounts for the infrequency of pneumothorax in this condition.

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GROSS PATHOLOGY OF INFLUENZAL PNEUMONIA IN FRANCE.

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THE material for this paper was collected from 106 autopsies on cases of pneumonia dying at a base hospital in France (Commercy,

Meuse). A few deaths occurred in December, 1918, the number increasing in January, reaching its highest point during the first two weeks in February and then gradually subsiding with scattered cases during March and April.

Only primary pneumonias have been included in this series and not pneumonias complicating typhoid fever, meningitis, traumata, etc.

PATHOLOGY. *General.* Of the skin manifestations, jaundice was the most common, occurring in 40 cases. Usually slight and only sufficient to stain the scleræ, the skin of the face and perhaps the aorta, it was not infrequently deep enough to stain the subcutaneous and mediastinal tissues and the kidneys. Herpes of the lips appeared 8 times, 3 times with lobar pneumonia, 5 times with lobular pneumonia. Skin or conjunctival petechiæ were never seen. A well-demarcated erythema of the tip of the nose, without swelling or edema occurred once. It was first noted one day before death.

Emphysema of the subcutaneous tissues of the neck, chest and shoulders occurred four times. The gas that rushed forth on opening into such an emphysematous area was odorless and non-inflammable. This condition was usually associated with emphysema of the soft areolar tissues of the anterior mediastinum. Occasionally blebs were observed between the deeper layers of fascia covering the chest wall and beneath the pleura. None of these cases, however, was associated with hemolytic staining.

Palpable enlargement of the axillary lymph nodes, especially on the right side, occurred 12 times. The nodes were removed and examined in 2 cases but aside from enlargement showed no gross changes.

Changes in the recti muscles occurred in 35 cases. At times merely an unusual pallor, dryness and opacity were present. Such muscles seemed to have lost their elasticity, were brittle and very easily fragmented and torn. Though this condition was usually diffuse, uniformly involving a considerable portion of the muscle, at times small islands of diseased muscle, rather sharply demarcated from the surrounding normal tissue, were seen. Usually, hemorrhage into the muscle, with rupture of the fibers and hemorrhage between the muscle and its anterior and posterior sheaths, were also present. The hemorrhage frequently dissected down into the space in front of the bladder and could often be seen through the peritoneum as a blue mass bulging into the peritoneal cavity. In one case complete rupture of both recti was found. The torn ends of the muscles were separated a full finger's breadth. This peculiar degeneration of the recti usually involves the lower fourth of the muscle, although similar changes are often seen, either alone or together with change below, in the recti at their attachments to the ribs. Abscess never occurred.

An edema of the right arm, with slight reddening of the posterior surface of the forearm, occurred once. The arm veins were dissected but no thrombi found. In this case the axillary and cervical lymph nodes on the right side were considerably enlarged.

Purulent inflammation of the right sternoclavicular articulation and of one of the lower cervical intervertebral articulations was found once, complicating a case of lobular pneumonia, with multiple lung abscesses. Splenic culture was sterile, cultures of both lungs showed Bacillus influenzæ, culture of the sphenoid and a pneumococcus.

Respiratory System. By far the most usual lesion encountered in the lungs was acute lobular pneumonia. The lungs were large, heavy and moist. On section one found, scattered throughout all the lobes usually, raised, granular, firm, grayish or red areas, about 5 to 10 mm. in diameter. The intervening lung tissue was red, moist and emphysematous. Occasionally dark red hemorrhagic areas, without consolidation, were seen. In one case intense congestion and edema of both lungs (from which the Streptococcus hemolyticus was grown in pure culture) was the only pulmonary lesion. The raised, granular areas were frequently confluent, especially in the lower lobes.

The next most frequent lesion met with in the lungs was lobar pneumonia. This differed from the lobar pneumonia met with in civil life in its bacteriology, streptococci being found in the lungs nearly as frequently as pneumococci, and in its not infrequent combination with lobular pneumonia.

Another type of pneumonia encountered occasionally (7 cases) was interstitial bronchopneumonia. These cases differed from those described by MacCallum in their bacteriology. One of the cases showed Gram-negative cocci in pure culture from both lungs and another showed staphylococci.

Empyema, as the predominating lesion, was found twice, serofibrinous pleurisy as the predominating lesion once, acute congestion of the lungs once and multiple abscesses once. Empyema, or purulent pleurisy, occurred but 4 times in the entire series. It was twice associated with multiple lung abscesses. Multiple lung abscesses were found 3 times.

The pleural cavities usually contained a few cubic centimeters of fluid. Hemorrhages into the pleuræ, both large and small, were very frequent. Fibrinous pleurisy occurred 28 times, serofibrinous pleurisy 12 times and empyema 4 times. The pleural fluid was frequently blood-tinged. The fibrinous exudate was usually small in amount, often merely an opacity on the surface of the lung. It was usually seen where the diseased lung tissue reached surface.

Bronchiectasis occurred three times, twice in the right upper lobe, once in the left lower lobe. Acute bronchitis was a very frequent accompaniment of the disease. It usually extended up

into the trachea, involving its lower half and fading out in its upper half. Purulent bronchitis was seen 5 times. Enlarged, soft, moist, reddish bronchial lymph nodes were found in 59 cases.

The larynx was examined in 38 cases. Congestion of the vocal cords (acute laryngitis) was found 6 times and edema of the epiglottis and arytenoepiglottidean folds twice. Occasionally hemorrhages into the mucosa covering the posterior surface of the epiglottis or thyroid cartilage were seen. Pharyngeal congestion was seen in a few cases. It was associated with a reddening of the base of the tongue and was sharply limited below at the commencement of the esophagus.

Circulatory System. Serofibrinous pericarditis occurred 6 times, purulent pericarditis once. Generally neither the quantity of fibrin nor fluid was abundant. It was always accompanied by acute pleurisy. The pericardium showed petechial hemorrhages in 49 cases.

Dilation of the heart was found in 43 cases, 31 times on the right side, 12 times on the left. The cardiac dilation was usually slight, the right ventricle perhaps occupying one-half the posterior surface of the heart instead of one-third, as it normally does. But occasionally it was marked and was twice associated with sudden death.

Cloudy swelling of the myocardium was found in 51 cases. The heart muscle was pale, soft and opaque. Occasionally a real flabby, thinned-out heart wall was seen, but this was rare. Acute vegetative endocarditis was found 4 times. The vegetations were small, always on the mitral valve, twice on chronically diseased valves, twice on otherwise normal valves. Hemorrhages into the cusps occurred in 7 cases, usually into the mitral valve, occasionally into the tricuspid and aortic valves.

In 3 cases the intima of the aorta was swollen, sticky and easily detachable (acute aortitis). Atheroma of the aorta and coronaries was noted in almost every case. It was, however, found with equal frequency in patients dying from other acute infections and from traumata.

Hemolytic staining of the endocardium or pericardium or both was observed 5 times. Most of these cases were autopsied twenty-four hours or more after death, although in one case autopsy nine hours after death revealed slight hemolytic staining of the endocardium.

Liver. Cloudy swelling of the liver was found in 85 cases, congestion 9 times. The cloudy swelling was usually very marked.

Spleen. Large, soft spleen was observed in 65 cases. The enlargement, as a rule, was not marked; the spleen was usually reddish and the Malpighian bodies were usually indistinct.

Genito-urinary System. Cloudy swelling of the kidneys was found in almost every case. It was usually well-marked. Renal congestion was also frequent, occurring in 37 cases. Acute nephritis

occurred 4 times, miliary abscesses of the kidneys once. Bile-staining of the kidneys in cases of deep jaundice was frequent. Microscopic sections of the kidneys in 3 cases, chosen at random from those showing cloudy swelling grossly, showed necrosis and desquamation of the tubular cells (convoluted tubules mainly), duplication of the tubular epithelium with hemorrhage into the tubular lumen, apparently a true acute tubular nephritis. The glomeruli, aside from congestion, were normal.

Hemorrhages into the renal pelvis occurred in nearly 50 per cent. of the autopsies (52 cases). The hemorrhages were usually small, but occasionally they were quite large, especially in the calices beneath the tips of the pyramids. The hemorrhages extended for a short distance into the ureter, but were never seen below the upper fourth. Hemorrhages into the bladder wall were noted 6 times.

Occasionally, on opening a testis, bloody fluid was seen. The prostate showed no changes.

Adrenals. Cloudy swelling of the adrenal cortex was seen in 23 cases. Congestion of the medullary substance was frequent. Hemorrhage into the medulla occurred 3 times. It was bilateral once, unilateral twice.

Thyroid. The thyroid was examined in 38 cases. Cloudy swelling was found 12 times.

Gastro-intestinal Tract. The gastro-intestinal tract showed few changes. Acute fibrinous peritonitis was found once. In this case the peritoneum covering the lower ileum was reddened, hemorrhagic and covered by a fibrinous exudate. The solitary lymph follicles and Peyer's patches were raised and reddened. Bloody intestinal contents were found once. Prominence of Peyer's patches and solitary lymph follicles, especially the latter, with or without reddening, were frequently noted in cases in which no other evidence of status lymphaticus was found. Acute appendicitis occurred once.

The cranial contents were examined in 26 cases. Meningitis was never seen. Cases of meningococcic meningitis, complicated by pneumonia, have not been included in this series. One case of pneumococcus Type I meningitis came to autopsy. The lungs showed an early lobular pneumonia of the left lobe. Aside from a very delicate tegmen tympani on the right side the search for an explanation of the infection was negative. The middle ears and sinuses were clear and the blood culture at autopsy sterile.

The brain often showed flattening of the convolutions and narrowing of the ventricles.

The sphenoid sinus was opened 22 times. Purulent fluid was found 18 times, serous fluid twice and in 2 cases no change was observed. Purulent frontal sinusitis and purulent otitis media were seen frequently, but no note of the number of examinations was made.

RESULT OF POSTMORTEM CULTURES.

Predominating organisms.	Number of cases.		Percentage of cases.
Streptococcus hemolyticus	18		32.0
Pneumococcus:			
Type I	3	5.2	
Type II	2	3.5	
Type III	1	1.7	
Type IV	4	7.0	
Not typed	6	10.5	
	16		28.0
Staphylococcus	9		15.5
Non-hemolytic streptococcus	5		9.0
Bacillus influenzæ	5		8.5
Gram-negative cocci	4		7.0
Total	57		100.0

BACTERIOLOGICAL NOTE. Thirty-five cultures were made from the circulating blood taken from the arm vein in the usual way. Of these 3 showed the pneumococcus Type I, 2 the pneumococcus Type IV, 2 pneumococci type undetermined and 1 the meningococcus. In 2 cases of lobar pneumonia, in which pneumococci were isolated from the blood during life, postmortem cultures from the lungs yielded the Streptococcus hemolyticus. It is probable that in these 2 cases at least the Streptococcus hemolyticus was a secondary invader.

The results of the postmortem cultures are shown in the accompanying chart. The Gram-negative cocci, which were found alone and frequently together with other organisms, resembled meningococci both morphologically and culturally, but they failed to agglutinate with polyvalent automeningococcus serum. Cultures from the sphenoid sinus gave very satisfactory results, the organism found there generally corresponding to the organism found in the lungs or blood.[1]

I wish to express my thanks to Dr. J. J. O'Leary, Detroit, who did a considerable part of the postmortem bacteriology.

SUMMARY. 1. During January and February, 1919, an epidemic of severe lobular pneumonia occurred about the region of Commercy, France, accompanied by jaundice, cloudy swelling of the parenchymatous organs and heart muscle and hemorrhages into the pericardium, renal pelvis and other viscera.

2. Changes in the rectus muscle were observed in 33 per cent. of the cases.

[1] The case in which the positive meningococcus blood culture was obtained, was clinically lobular pneumonia. There were no signs of meningitis and a lumbar puncture did not seem advisable. The patient recovered. The organism was grown in plain infusion broth to which the blood had been added after five days' incubation. The organism was typical morphologically and culturally and was agglutinated by polyvalent antimeningococcus serum, dilution 1 to 200 and failed to agglutinate in normal sera. It was not typed.

GROSS PATHOLOGICAL FINDINGS.

	Number of cases.	Percentage of incidence.
Number of autopsies	106	
Jaundice	40	30.0
Herpes	8	7.5
Subcutaneous emphysema	4	3.7
Degeneration of recti	35	33.0
Edema of arm	1	0.9
Purulent arthritis	1	0.9
Enlargement of axillary lymph nodes	12	11.3
Respiratory system:		
Lobular pneumonia	77	72.6
Lobar pneumonia	14	13.0
Interstitial pneumonia	6	5.6
Lobar and lobular pneumonia	5	4.7
Interstitial and lobular pneumonia	1	0.9
Multiple lung abscesses	3	2.8
Fibrinous pleurisy	28	26.2
Serofibrinous pleurisy	12	11.3
Empyema	4	3.7
Bronchiectases	3	2.8
Acute bronchitis	82	77.0
Purulent bronchitis	5	4.7
Large, soft bronchial lymph nodes	59	55.0
Number of larynges examined	38	
Acute laryngitis	6	15.7
Edema of glottis	2	5.2
Heart:		
Serofibrinous pericarditis	6	5.6
Purulent pericarditis	1	0.9
Petechial hemorrhages in pericardium	49	46.2
Cloudy swelling of heart muscle	51	48.0
Cardiac dilatation—right	31	29.0
Cardiac dilatation—left	12	11.3
Acute endocarditis	4	3.7
Hemorrhage into cusps	7	6.6
Hemolytic staining of endocardium	5	4.7
Acute aortitis	3	2.8
Liver:		
Cloudy swelling	85	80.0
Congestion	9	8.4
Large, soft spleen	65	61.0
Kidneys:		
Cloudy swelling	93	87.0
Congestion	37	34.8
Acute nephritis	4	3.7
Miliary abscesses	1	0.9
Hemorrhages into renal pelvis	52	49.0
Hemorrhages into bladder wall	6	5.6
Adrenal:		
Cloudy swelling	23	21.6
Hemorrhage	3	2.8
Thyroid:		
Number of times examined	38	
Cloudy swelling	12	31.5
Gastro-intestinal tract:		
Fibrinous peritonitis	1	0.9
Sphenoid sinus:		
Number of cases examined	22	
Serous sinusitis	2	9.0
Purulent sinusitis	18	81.8

3. Empyema was rare, occurring in less than 4 per cent. of the cases.

4. Acute laryngitis was comparatively rare, occurring in but 6 out of 38 larynges examined (16 per cent.).

5. Sphenoid sinusitis was a very common complication and was found in 20 out of 22 cases examined (90 per cent.).

6. Seven out of 35 premortem blood cultures showed pneumococci. The rest were sterile with the exception of one, which showed a meningococcus.

7. The bacteria found in the organs at autopsy were varied, the Streptococcus hemolyticus, pneumococcus, staphylococcus, nonhemolytic streptococcus, Bacillus influenzæ and Gram-negative cocci being found in the various cases in the order of frequently given above.

CHRONIC NON-TUBERCULOUS LUNG INFECTION.

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The material for this paper consists of eight cases from a somewhat larger number seen during the past year in the medical clinic of the University Hospital of the State University of Iowa. Our object has been simply to review the literature and to emphasize some points in the diagnosis and treatment of this condition which so closely resembles tuberculosis and which is so frequently erroneously diagnosed as such. We do not claim any originality either in diagnosis or treatment, both of which have previously been fully described.

The recent influenza epidemic left in its wake countless cases of chronic cough, many of which are unquestionably due to tuberculosis, as is proved by the finding of acid-fast bacilli in the sputum. However, in many so diagnosed tubercle bacilli are not found and the subsequent course is unlike tuberculosis. Many of these cases are simple bronchitis, general or localized, or, to use the term recently suggested by Garvin, Lyall and Morita,[1] chronic nontuberculous lung infection.

In 1902 and 1905 F. T. Lord[2] described a series of these cases under the title of chronic influenza. Since then similar cases have been reported by Riesman[3] in 1913, Larabee[4] in 1915 and more recently papers on the subject have appeared by Hammond and

[1] Am. Rev. Tuberculosis, 1917, i, 16.
[2] Boston Med. and Surg. Jour., 1902, cxlvii, 662; Boston Med. and Surg. Jour., 1905, cliii, 537.
[3] Am. Jour. Med. Sc., 1913, cxlvi, 313.
[4] Boston Med. and Surg. Jour., 1915, clxxii, 257.

Wollman,[5] Garvin, Lyall and Morita and J. A. Miller.[6] The disease has been variously called "a lobular form of bronchopneumonia of long duration," "localized bronchitis," and "subacute and chronic non-tuberculous lung infection."

ETIOLOGY. *Age.* The onset may occur at any age, but seems to occur somewhat more frequently in children. G. F. Still,[7] who has described this disease in children, says it is most frequent before the age of five, due not only to the frequency of measles and whooping-cough, which diseases it often follows, but because of a special predisposition of the bronchi of children to infection. In children there is a relatively large amount of loose connective tissue in the walls of the bronchi and alveoli, which is easily infected and permits chronic dilatation of the bloodvessels to occur with relative ease. The frequency of mouth-breathing and chronic nasal disease in children also undoubtedly increases their liability to bronchial disease. Of our 8 cases, 3 began before the age of six, 2 at thirteen and 3 at twenty. Miller's cases ranged from seven to sixty years. The majority of Riesman's cases were between ten and twenty.

Sex. The disease is more common in females. In Miller's series there were 14 females to 8 males. In Riesman's series girls predominated. Our cases were divided equally between the two sexes.

Previous Infection. The syndrome usually follows acute diseases, which affect particularly the upper respiratory tract, such as measles, whooping-cough and influenza, though in some cases it comes on insidiously.

Season. Both the onset and the exacerbations occur more frequently during the colder months, October to April, due probably to the crowding of people together in poorly ventilated and often dusty or smoky rooms and the increased tendency for respiratory disease to pass from person to person under such circumstances.

Nasal Disease. Adenoids and nasal deformities which cause mouth-breathing are important etiological factors. Chronic sinusitis is probably present to a greater or less extent in all the cases. Three of our cases had definite severe sinus diseases and in all the lung exacerbations were invariably accompanied by coryza.

Family. Miller reports 2 cases from the same family in two instances. In each instance the same organism was isolated from the 2 cases, suggesting the possibility of contagion. Two of our cases came from the same home and Staphylococcus albus was isolated from the sputum of each of them. The same predisposing factor, as poor ventilation, with dust or smoke, may help in increasing the family incidence.

Deformed Chest. Still mentions the frequency with which localized bronchitis may occur in rachitic chests, due to the fact that

[5] Tr. Nat. Assn. Study and Prev. Tuberculosis, 1916, p. 171.
[6] AM. JOUR. MED. SC., 1917, cliv, 805.
[7] Common Disorders and Diseases of Childhood, p. 353.

portions of the lung cannot properly expand and easily become chronically infected. None of the cases here reported had any deformity of the chest whatever, but we have seen some cases in which the deformity seemed to be of etiological significance.

Bacteriology. Bacillus influenzæ is the most frequent predominating organism in the sputum and was found in 36 per cent. of Lord's cases, but any of the common invaders of the respiratory tract may be found. In our 8 cases Bacillus influenzæ was found to predominate in only one instance, while the Streptococcus pyogenes was predominant in 3 and Staphylococcus albus in 4. In one of the streptococcus cases a similar organism was isolated from pus found in the antrum of Highmore. Miller's cases showed Bacillus influenzæ in 7, pneumococcus, type IV, in 10, and Streptococcus viridans in 4.

Out of 66 cases Lord found Bacillus influenzæ in 44, pneumococcus in 8, Micrococcus catarrhalis in 5, Bacillus mucosus capsulatus and pseudopneumococcus in 3 each. A different organism is sometimes found to predominate at a later time, as was the case in one of our patients who had Bacillus influenzæ at first; but two weeks later Staphylococcus albus was found to be the predominating organism. Lord found such a change to occur in some of his cases.

Pathology. Since this is a relatively benign process there has been very little opportunity to make postmortem examinations. None of our cases came to autopsy. Hammond and Wollman had an autopsy on one case and found "localized bronchitis and infiltration of the bronchial wall and foci of bronchopneumonia about the smaller bronchi." Lord examined, postmortem, a typical case of forty-four years' duration and found slight general bronchiectasis, with many small areas of interstitial pneumonia. It therefore seems probable that, as Miller has stated, the pathology "may consist of a localized bronchitis, with a lobular distribution, which may clear up or persist in a subacute form, offering a site predisposed to exacerbations and increased tendency to fibrosis." That the late changes are bronchiectasis and areas of interstitial pneumonia is shown by Lord's case. The tendency in some of the cases for the cough to become paroxysmal, with considerable amounts of sputum, shows there must be some dilatation of the smaller bronchi fairly early in the course of the disease. The roentgenograms often show some fibrous thickening, which occasionally pulls the heart slightly to the affected side.

Symptoms and Course. Chronic cough, usually most marked in the mornings, and expectoration of variable amounts of mucopurulent sputum are the outstanding features in practically all the cases. The general health is relatively little affected, but many of the cases complain of slight loss of appetite, slight loss of weight and lack of endurance. Four of our cases complained of these, but the other four claimed they were in perfect health except

for cough and expectoration. One of our cases had several frank hemorrhages, two of which consisted of as much as a pint of bright red blood. Many cases with hemoptysis have been reported in the literature. Moderate fever, night-sweats, pain in the chest, in short, any of the symptoms of tuberculosis may be present. Riesman has pointed out that subacute and chronic lung infections may be the cause of obscure long-continued fever, as some of these cases complain of no symptom referable to the chest, and it is only by making routine chest examinations and finding the typical signs that the nature of the process is suspected.

The course of the disease is marked by extreme chronicity, with a tendency to frequent exacerbations. The latter are usually accompanied by coryza, fever, increased cough and tightness in the chest, frequently pain in the affected side, and occasionally, as in one of Miller's cases, slight effusion. Some of the cases go on to complete recovery in from two to six months, but when the condition has become well established the patients go on from year to year with nothing but temporary abatements, and eventually the general health becomes more or less impaired. The prognosis is best in children.

Physical Signs. The site of the lesion in our 8 cases was the left lobe in 5, the right lower lobe in 1 and both lower lobes in 2. In 7 of Lord's chronic cases the right lower was affected in 2, the left lower in 1 and one or both apices in 3. Garvin, Lyall and Morita state that in 90 per cent. of the cases the lesion is in the lower half of the lungs. Inspection reveals nothing abnormal, or, at most, only slight lagging of the affected side. The typical signs are subcrepitant rales, especially after cough, in one of the lower lobes. These may be brought out by placing the patient in the inverted position, as has been emphasized by Garvin, Lyall and Morita. Over the affected area the percussion note is often somewhat impaired and the breath sounds somewhat harsh. Physical signs are frequently insignificant and occasionally entirely absent.

Three of our patients were rather pale, emaciated and "sick" looking, but all the rest were well nourished and of good color. Two had considerable incurvature of the finger nails. All had slight leukocytosis (11,000 to 12,400 per c.mm.) and three had slight anemia.

All of our cases raised mucopurulent sputum varying in amount from ½ dram to 1 ounce daily. Two cases were not expectorating anything when admitted, but upon being placed in the inverted posture for fifteen minutes, three times a day, both began bringing up small amounts of mucopurulent sputum. The sputum in most of our cases contained the green nummular masses so often seen in influenza. Tubercle bacilli were never found in any of the sputa after repeated examinations both with and without antiformin.

The von Pirquet test was negative in the four children. Two of

the adult cases gave no general or focal reaction to the subcutaneous test. The latter test was not tried on the other two adult cases.

DIAGNOSIS. That the diagnosis is frequently missed is shown by the fact that one-half of our cases were previously diagnosed tuberculosis. Cases with chronic cough, which is not paroxysmal; purulent sputum persistently negative for acid-fast bacilli; insignificant roentgen-ray findings, with rales located in the lower half of the chest (in the absence of a heart lesion); and the general health only slightly impaired are undoubtedly cases of chronic non-tuberculous lung infection.

When the lesion is located at one or both apices the diagnosis is more difficult and tuberculosis cannot always be positively excluded even after prolonged observation. Against the latter, however, is the fact that the disease has no tendency to spread to other parts of the lung and the sputum never contains tubercle bacilli. The absence of acid-fast bacilli from purulent sputum, after careful and persistent search, can be said, for all practical purposes, to rule out tuberculosis. The general robustness of the patients in spite of the extreme chronicity of the disease and the frequent exacerbations, the negative roentgen-ray finding and negative tuberculin test are sufficient evidence to exclude tuberculosis.

In our 4 cases which were previously diagnosed tuberculosis the signs were entirely confined to the lower half of the lungs. Basal lesions in tuberculosis are rare and are always toxic. The good, general health of these patients, in relation to their rather extensive physical signs of lung involvement, made us doubt the diagnosis at once.

Subacute cases, with moderate daily rise of temperature, may at first resemble typhoid fever; but the slight leukocytosis, negative Widal and blood culture, together with the finding of the characteristic lung signs, should suggest the correct diagnosis.

Bronchiectasis can usually be excluded by the fact that the sputum is brought up in smaller amounts and not in the paroxysmal manner so characteristic of that disease. However, as mentioned before, sooner or later some of the cases probably develop slight bronchiectasis.

Sometimes the disease may be recognized at its very beginning by careful examination before discharge of all cases of influenza, acute bronchitis, measles, etc. Recently one of our patients with acute bronchitis, who had never been troubled with cough before, was about to be discharged after being free from temperature and symptoms for several days. On routine examination of the chest the left lower lobe was found to be filled with crackling rales, and the percussion note was somewhat impaired over this area.

During the six weeks since he has been reporting regularly to the out-patient clinic, and, in spite of treatment, the signs still persist, and he has now considerable cough and expectoration.

TREATMENT. As Garvin, Lyall and Morita have pointed out the essential treatment is posture. The constant hacking which these patients carry on never entirely frees the smaller bronchi from secretion and alleviates the desire to cough for only a brief period of time. Drainage can be accomplished by having the patient hang over the end of the bed or kneel on a chair, with his hands on the floor. Placing the patient in the Trendelenburg or reverse Trendelenburg position on an operating or treatment table is more comfortable for the patient, and in some cases produces better drainage. The posture should vary somewhat, depending upon which part of the lung is to be drained. It may be necessary to try several different positions before the one which accomplishes adequate drainage is determined. The experience of the patient is often valuable in making this determination. The treatments should be of sufficient length and frequency to keep the affected area fairly free from secretion. Fifteen minutes, three or four times a day, are usually necessary at first. All of our cases have received distinct benefit from this form of treatment. Patients who have been coughing more or less all day long, and even having their sleep disturbed by paroxyms of coughing, soon cough only at the time of inversion. The amount of sputum gradually becomes less and the general health rapidly improves. There may be a slight febrile reaction after the initial drainage, but after that the toxic symptoms rapidly disappear entirely. Two of our cases who were raising no sputum began to expectorate from 1 to 4 c.c. daily after postural treatment was started, thus giving us an opportunity to examine the sputum in addition to putting an end to their cough and afternoon fever. During or immediately after inversion the rales can be much more easily heard, and, in fact, may be heard only at this time.

It may be necessary to continue this inversion over long periods, a year or more, but patients soon become accustomed to it, and the benefit is so obvious that they are usually enthusiastic and can readily be persuaded to carry it on in their homes. Our patients also received inhalations of compound tincture of benzoin following the inversions. In view of the fact that the affected part of the lung may be held down by adhesions, pulmonary gymnastics in the form of blowing-bottles should benefit some of the cases. This has been tried, with considerable success, in this hospital in the department of pediatrics, where a number of these cases have occurred. In all the cases the nasal sinuses should be carefully examined and any existing disease eradicated as far as possible.

Since the exacerbations occur in the colder months it may be advisable to recommend a warm climate for the chronic cases that are refractory to treatment. Exacerbations should be avoided by living an out-of-door life and avoiding exposure to people suffering from acute colds. Irritants to the bronchial mucosa, such as tobacco smoke and dust, should also be avoided.

We used an autogenous vaccine in 2 cases, but noticed no more improvement than in similar cases which were receiving only postural treatment.

I beg to acknowledge my appreciation to Professor C. P. Howard for giving me the opportunity to study and report these cases.

CASE REPORTS.

E. S., No. 6275. White boy, aged fourteen years, entered May 19, complaining of cough and expectoration dating from measles at five. He was well nourished and there were subcrepitant rales in both lower backs as high as the angles of the scapulæ. The sputum was negative for tubercle bacilli and the cultures showed a marked predominance of the Streptococcus pyogenes. Afternoon temperature, 99.6° F.; leukocytes, 11,000; Wassermann, von Pirquet and roentgenograms negative; chronic empyema of antrum of Highmore. Antrum was drained and reverse Trendelenburg posture for fifteen minutes, three times a day, were given. Discharged June 20, free from cough, expectoration and temperature.

O. M., No. 6337. Male, aged twenty-two years, entered June 4, with headache, cough and temperature of 102.8°, which began the day before. Past pulmonary history negative. Lungs negative. Temperature became normal on the sixth day but continued to raise large amounts of green nummular sputum, slightly streaked with blood. At this time the left lower lobe was found to be filled with subcrepitant rales. Six weeks later, when seen in the outpatient clinic, the signs were unchanged and the sputum contained a pure culture of Bacillus influenzæ. He is now rapidly improving on postural treatment.

E. C., No. 6274. White male, aged nineteen years, entered May 19, complaining of constant cough and expectoration dating to whooping-cough, two years ago. During the last four months he had lost ten pounds and felt weak and tired. He was pale and emaciated. The lung signs were sharply localized to the right lower lobe, where the percussion note was impaired and many subcrepitant rales were heard. The sputum showed Streptococcus pyogenes and no tubercle bacilli. Afternoon temperature, 99.2° F.; leukocytes, 11,000; von Pirquet and roentgen rays negative. Carried out postural treatment at home. Two months later cough and expectoration had ceased and general health was much improved.

A. De S., No. 6114. White male, aged twenty-six years, entered April 3, 1919. Had constant cough, with frequent exacerbations and variable amounts of thick, yellow sputum as long as he could remember. Color and nutrition were normal and both lower lobes filled with rales. He was raising about one ounce of sputum daily, which contained a predominance of Staphylococcus albus. Afternoon temperature, 99.2° F., Wassermann, von Pirquet and roent-

genograms were negative. Chronic pansinusitis. Drainage of the sinuses and postural treatment for one month resulted in marked improvement in the cough and reduction of the daily amount of sputum to ½ c.c.

E. B., No. 5810. White girl, aged fifteen years, entered January 10, 1919. Had been coughing since measles at six. Expectorated one dram to an ounce of sputum daily. Color and nutrition normal. Both lower lobes were filled with rales brought out by coughing. Afternoon temperature, 99.6°; leukocytes, 12,200; red cells, 3,910,000; hemoglobin, 75 per cent. Sputum was green nummular type and cultures showed Staphylococcus albus. Wassermann and von Pirquet negative. Roentgen rays showed considerable fine radiating thickenings at the bases. Sinuses negative. After two months of postural treatment the anemia had disappeared and she had gained six and three-quarter pounds. Cough, sputum and lung signs had disappeared.

L. C., No. 4816. White female, aged fourteen years, entered June 16, 1918. Had a chronic cough since whooping-cough in July, 1917, which had become much worse since she had measles in May, 1918, since when she has expectorated about one ounce of sputum daily, perspired profusely at night and lost weight and strength. She was very pale and emaciated and there was slight clubbing of the fingers. There was impaired percussion note and numerous moist rales over the left lower lobe. The sputum contained Streptococcus pyogenes. Roentgen rays, von Pirquet and Wassermann negative. Afternoon temperature, 99.2°; leukocytes, 11,000. Was discharged July 25, 1918, free from cough and expectoration. She had gained six pounds; color and temperature were normal.

L. E., No. 6299. White female, aged twenty-seven years, entered May 24, 1919. Had a severe cold eight years ago, during which she had several hemoptyses, two of which were as much as a pint. Has had constant cough since. Sputum is often blood-streaked. She looked healthy and the only signs found were impaired percussion note and moist rales over the left lower lobe. She was not expectorating when admitted, but after postural treatment was begun she raised one dram daily of green nummular sputum which contained Staphylococcus albus, and the cough ceased except at time of inversion. Subcutaneous tuberculin test and roentgenograms were negative.

E. D., No. 6374. White boy, aged thirteen years, entered June 16, 1919. Had whooping-cough at nine and has since had cough and expectoration, with frequent exacerbations. He was pale and emaciated and running an afternoon temperature of 99.8° The lower left lobe was filled with rales. Leukocytes, 12,000. Von Pirquet and roentgen rays negative. Sputum contained Staphylococcus albus in almost pure culture. Temperature and weight became normal and cough and expectoration much improved after two weeks.

A PSYCHOLOGICAL THEORY OF THE CAUSE OF EPILEPSY, WITH SPECIAL REFERENCE TO AN ABNORMAL MUSCULAR EXPRESSION OF A STRONG EMOTIONAL DRIVE.

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Epilepsy, though it has existed and has been studied as long as we have written history, is so complex in its phenomena that it has baffled the medical profession in establishing its etiology. It is now broadly defined as a chronic progressive disorder characterized by recurrent abrupt attacks of loss or impairment of consciousness with or without convulsions and accompanied ofttimes by a physical and by a mental deterioration. Recent medical investigation, since there is no definite pathology known common in every such case, has been an attempt to study the disease process chiefly from the viewpoint of a mental disorder.

In every individual case of epilepsy we find certain peculiar but definite mental characteristics which are common to all so afflicted. The mind of the epileptic is filled with thoughts and feelings of an unpleasant nature, so that he is seldom if ever happy. He is constantly wanting something that he cannot get. In his efforts to gain the thing desired, he possesses a strong determination that is characteristic only of his type of mentality. Although physically and mentally he is entirely incapable of attaining the things he desires, he possesses an unreasonable confidence in his own ability to overcome any obstacle. Failing to do so, he becomes temperamentally a misfit wherever found. He is sensitive about his incapability, which he will not recognize, is apt to be irritable when approached and becomes a patient subject to tantrums and even acts of violence when not managed with the greatest degree of caution and understanding. Wrapped up in his likes and dislikes of the unobtainable, he can usually be expected to miss the pleasure that he might find in performing tasks within the scope of his capabilities. This mental make-up, characterized by supersensitiveness, egocentricity and emotional poverty, does not help the epileptic to make proper adaptations in life.

We might conclude that given an individual with such an emotional constitution plus a particular type of stress we should get a particular epileptic reaction. Such a generalization, while it may be true, leads us into conditions difficult to explain. Every person, to a greater or lesser degree, manifests these same mental peculiarities and also is subjected to most every form of stress, so we shall have, then, to consider everyone, both young and old, as potential epileptics. The surgeon may, though he probably looks

forward with hopes to the time when he will not be asked to operate to relieve epilepsy upon clinical advice of such a doubtful nature as is given today, want more definite proof that the phenomenon is purely an abnormal mental reaction. Perhaps also we should consider that the many theories advanced by the internist, based on subjects as yet not well understood, such as endocrinic disturbances, intestinal intoxication, acidosis and increased intracranial pressure, may have an element of importance, since they probably deal with a resultant abnormal physiological process. These are steps made in the right direction and, like institutions, they are neither made nor destroyed in a day but must stand until supplanted by something better. Therefore, we must have here more than a generalization. We must, by a proper analysis, show the particular factors in a mental process that produces an epileptic reaction and show how it may be avoided.

A proper analysis of a mental, diseased state presupposes a working knowledge of normal mental activity. This is just as true as it is in the field of pathology, where we must first know normal tissue before we study the abnormal. Likewise, we study the physiology of the normal body before we investigate abnormal physiological processes. So any study of this subject, if it be of import, must show how the mental life of the epileptic differs from the mental life of a normal individual and consequently brings about his destruction.

Mental life is primarily teleological. Our inner faculties, that is, our instinct and ways of thinking, feeling or desiring, come to us when things interest or excite us, and they act as the motive power, which, when directed in natural channels, serve to secure our common welfare and safety. These feelings may, however, when improperly directed, find expression in an abnormal manner, and if habitually exercised in this way, may lead to the possessor's destruction.

Every mental state of this kind has a muscular expression. We recognize them in the voluntary and involuntary muscular reactions. We have, as an example of the latter, inconspicuous changes in breathing, in circulation, in general muscular tension and visceral changes, brought on by various emotional states. A definite feeling of this kind, which drives both voluntary and involuntary muscles to action, is hunger. We get hungry, go about getting something to eat and then find pleasure in eating as our hunger is satisfied. So these feelings, whether we are conscious of them or not, produce muscular activity which is brought about to meet some particular adaptation essential to the body's welfare.

Feelings which stir up voluntary muscular reactions are apt to be more keenly felt. Danger, for example, fills us with fear and the natural expression of this emotion is to exert our muscles in such a manner that we shall escape it. Flight is quite often the usual action

taken. If by running away we escape the danger, we experience a sense of pleasure in our success. While running, though, should we feel a doubt of our success in escaping the pursuing danger, our experience is very unpleasant, and the greater our doubt the greater is our fear, so we are able to run all the faster.

Another mental state which exemplifies a muscular expression of the voluntary type is seen in the reaction that follows anger. The natural expression of this emotion is to overcome the thing which causes the anger. Perhaps it means a fight. It is an interesting observation that, in a large, open field a man cannot fight as hard as he can when he is cornered. In the open he has plenty of room to maneuver, with the possibility of turning in flight if the battle grows too strong. Cornered, he has less space for manuever, with no chance of escape except by overcoming his adversary. In this case the emotional feeling which gives him motive power for his efforts is not weakened by conflicting thoughts, but is made stronger by the increase of impending danger.

We cannot consider here all of our thoughts and feelings or whatever other names we may give our mental states, for they are infinite in number. We can, however, from our knowledge of them, draw this general conclusion: that they are, whether we are conscious of them or not, the motive power behind our efforts to do or to get things for ourselves or for those in whom we are interested. With such motive force calling for expression, five different ways are found as an outlet for these feelings. When we want to do or get something we may be:

1. Successful.
2. Unsuccessful—suicide.
3. Unsuccessful—cease trying.
4. Unsuccessful—insanity.
5. Unsuccessful—epilepsy.

Probably no life is so unpleasant as never to experience success in some degree. Nevertheless, to be successful in everything one attempts is doubtless a greater pleasure than anyone has ever experienced. So we mention the first manner in which feelings may find expression only as an ideal never reached.

The second also has little interest for us except that we must accept it as a frequent outcome to ill-success. Suicide is an abnormal manner of escape, but one that places the one who considers life not worth living, if he cannot do or get the particular thing of sole interest to him, soon beyond burden to others.

In the third we have to consider the action of the normal man who meets unsurmountable obstacles. Wanting to do or get something, he puts forth every reasonable effort. As he ponders and studies, perhaps worrying a little more than he should over his troubles, seeking some new way of approach that he may be successful, he soon feels the repulsiveness of greater effort in the face of the

futility of any attempt, so he ends the unpleasant experience by turning to other tasks which he can accomplish and thus finds pleasure in their success. The instances in which the normal man gives up when demands become too trying are as many and as varied as there are interests in life.

The fourth way of meeting a difficulty is a manifestation seen in insanity. In this condition, often complicating epilepsy, we do not find an individual who has formed the habit of avoiding the danger of too great mental stress as the normal man meets it. Instead, the unnatural habit is made of escaping an intolerable situation of unsurmountable circumstances through a mental state made more tolerable in a world of imagination, where hallucinations, illusions and delusions play a make-believe part.

The fifth and final way in which the motive power of emotional life finds expression, when the natural pleasurable channel of success is blocked, is through an epileptic reaction. Possessed with a mental make-up characterized by egocentricity and an emotional poverty for feelings not particularly concerned in his immediate desires, the epileptic, like the man cornered, has no possibility of escape except to accomplish what he desires or succumb in his efforts. He could avoid the latter outcome if he did not possess such an exaggerated notion of his own ability, and like the normal man could give up trying. But he labors on with every obstacle serving to aggravate his emotional drive to the point of an abnormal explosion.

Fatigue is a natural consequence of any mental activity, and it demands rest which we get normally when we sleep at night. If, however, the mental work is of the nature of violent strain, such as is experienced in extreme emotional effort, an abnormal degree of fatigue or exhaustion is had which calls for an immediate cessation of function until a period of rest intervenes. This is what happens in the phenomena of epilepsy when the patient falls unconscious. The higher brain centers which have to do with the directing and with the consciousness of efforts become exhausted from overwork when subjected to extreme nervous tension. This loss of consciousness is not deep enough to involve the motor centers, so the emotion goes on to an abnormal expression in muscular activity, unguided and uncoördinated, which we know as a convulsive seizure.

Two cases of this kind, which, from all the evidence gathered from the patients and their friends who knew them in the army, were the first attacks suffered of the kind, came to my notice while in France. The first was that of an excellent soldier of the infantry, an Armenian with our army, who had difficulties with the first sergeant of his company. The sergeant, of German parentage, possessed many racial characteristics. Although a fine specimen of physical manhood and an excellent American soldier, his very

appearance was odious to the Armenian, who had lost his wife and children in the massacres of his native land. Because of these differences, the Armenian was detailed to the officers' mess for duty, where he would be less apt to come in contact with the first sergeant. Some time later, however, their quarrel was renewed, when the first sergeant, in language which betrayed not a long separation from that of the Fatherland, proceeded to give the Armenian a lecture on proper respect to the first sergeant of the company. Angered to the breaking-point, the Armenian would have crushed him had he dared to violate the laws of military discipline; but inhibited, he faced his aggressor until in emotional exhaustion he fell unconscious, and the emotion went on to an abnormal expression in a typical grand mal attack.

The second case was that of a sturdy soldier at the port of embarcation for home. Burning up with the desire of getting home, an emotional state of feeling, known only to those who have experienced it, the soldier met difficulties because of a lesion of a suspicious venereal nature. Having passed daily venereal inspection for more than one month, and knowing that he had not been exposed to infection, the mental shock, when he was informed by the inspecting surgeon that he would be held for further observation, was more than he could endure. He fell unconscious in a convulsion not unlike that of an epileptic seizure.

From such an analysis of mental activity we arrive not only at the fundamental factors causing the phenomena of epilepsy, but we come also into possession of knowledge which guides us in our efforts to combat the disorder. We have seen that all muscular activity is dependent upon a motive force which we are wont to call feelings, or emotional state of mind, and that these emotional states are intensified in the face of opposition, unless other feelings of an irrelevant nature, which tend to weaken them, are entertained. Consequently, every individual must acquire the habit of a broad interest in the things of life that he may the more readily escape the unpleasantness of failure in particular undertakings.

This is a normal habit of which we have countless examples. In the teachings of most of our institutions of learning, emphasis is laid upon the necessity of a broad education in general subjects instead of intensive work in particular branches. The experience of the race in this direction is exemplified by many instinctive actions that may be observed. A mother will not allow her baby to lie neglected in a fit of crying, but by fondling and caressing helps it to overcome its intense emotional state of unpleasantness. Neither will she allow it to be teased or otherwise encouraged in unpleasant feelings. Who has not heard a mother's admonition, "Don't frighten the baby; you will scare it into fits?" Babies are given toys and playthings that they may be pleasantly occupied.

In a kindergarten the trying ordeal of keeping a child pleasantly engaged is minimized through a teacher trained in the art of leadership. It is an interesting spectacle to see how such an instructor can keep, all the morning and afternoon, many children happy at play. Each disturber who threatens the contentment of all present has his attention taken from his own particular whims by the mere suggestion of a new game, and before it can be realized, all are happy once more, singing and playing without a harsh word given or anyone spanked to start all crying. It is necessary, also, to make the common and high-school work attractive, for the young resent the least overtaxing of their mental powers. In college a student is urged to take part in athletics, so as to relieve his pent-up energies. A business man finds hobbies, golf or fishing, a side line to take his mind from the worries of his office. These are protective influences which tend to weaken emotions which tax our mental strength.

Everyone feels these needs instinctively, yet every individual is conscious of an opposite tendency in this world where alone the strongest survive. Everyone feels the need of sharpening his interests down to particular ends. This may be done within certain limits, but when carried too far, brings on abnormal nervous conditions. A child which manifests an intensive application to study, although we may marvel at its accomplishments, is considered precocious, and we are fearful that it cannot withstand such mental effort. The genius, limiting his field of activity, applies his marvellous amount of energy in accomplishing results of astounding importance. Yet society looks upon him as eccentric, or even crazy, because of his poverty of broad interests. He is abnormal because most men of his type become failures in a nervous breakdown of some form.

The nature of our emotional life, then, is largely one of habit in our attitude toward things about us. So, confronted with the necessity of making difficult adaptations, we can expect the resultant reaction to be normal only when there is present a well-rounded emotional development. When, therefore, we see manifestations of emotional poverty we should be on our guard for abnormal expressions in particular emotional stress. A form of stress not usually looked upon as emotional, because the patient may not be conscious of any particular feeling, is had in acute febrile states, in toxemias and the like. Under such conditions the life of the individual is threatened. It does not make any difference what the form of attack may be, even though it be worms or even eyestrain, the body as a living organism resents any form of infringement on its welfare. Therefore, if consciousness is impaired there may be an abnormal expression of that power of resentment. A convulsion is one of the abnormal forms.

In anesthesia we produce, by means of ether as a drug, an attack

on the life of the body as a living organism. The higher centers of the cerebrum are first exhausted and cease to function. If care is not taken to gain the patient's confidence before these centers are put out he has a well-developed emotional state of fear because of the ordeal before him. Even though the patient may come to the hospital unconscious from trauma, by forcing the ether the air passages are strongly irritated, producing a special insult against the welfare of the body, which will be resisted even though the conscious centers of the brain are not functioning. In anesthesia the muscular action, as an expression of resentment, becomes what we call the excitement stage and it continues until the motor centers are paralyzed. These are all abnormal expressions of emotions, and once exercised become easier with each repetition. If we would guard against a habitual exercise of such abnormal expressions we must not only combat disease processes but we must so govern conduct that strong emotional activity will be avoided.

The same principles are involved in the care and treatment of a patient who has a well-developed disorder of epilepsy. The scope of this article does not permit a discussion of the details of the method of application of these principles, for each case is largely a matter for individual consideration; yet there are certain general features of the work which may be mentioned.

The hopes for improvement and recovery in every particular case of epilepsy lie in the patient's ability to become pleasantly occupied. Patients of low-grade mentality, patients past middle age and those in whom the process of epilepsy is long established in mental habits firmly fixed offer only a guarded prognosis. It is with the young and with those early in the process of the disorder that we expect favorable results. So we look forward to the time when, instead of the greater percentage of those received being chronic in their affliction, we may have more enter suitable institutions when their epileptic seizures are first manifested. Many patients are also handicapped with physical disabilities which have been a hindrance to them all through their life. Such deformities often serve as the exciting force which narrows the field of their pleasurable activities. When possible these physical defects are corrected and the patient is put in an environment where he is not always made conscious of his troubles.

To afford an opportunity where patients of this kind may work and not be constantly reminded of their affliction as they are when employed at home the State of Indiana has established this farm, through the efforts of a leadership of the caliber of constructive genius. The wisdom of the undertaking is seen in the improvement of a patient after coming here. Almost without exception every such individual not only gains in physical health by taking on weight but makes decided mental improvement, with the result that his seizures become less frequent. Many patients coming in shackles

from insane hospitals, where they are considered the most objectionable and difficult to manage of all patients there, are in a short time found peaceably at work at various tasks on the farm.

The problem, however, of changing the mental attitude of the epileptic does not end in providing him with an opportunity for work in which he may become interested. A person habitually self-centered is always ready to resent anything that will oppose his particular ideas. The epileptic is such a person. He cannot be ordered to do a thing or be forced to work and be expected to do it joyfully. Because of his mental attitude and resultant seizures, which he considers as an affliction, he sets himself up as an object to be pitied, but he will not be satisfied with any sympathy which may be shown him. Sympathy he does not want, and he will not stand abuse. A plain understanding is all that is necessary to satisfy him. With this he seldom fails to respond to any suggestion honestly and pleasantly given.

Such characteristics cannot be said to be wholly abnormal, for no person can will or be forced to will to do a thing. There must be a reason or desire to do strong enough to overcome any conflicting notion. To help them to have desires to do things, to place them in surroundings that will aid in this, is the big thing to be considered in the care and treatment of the epileptic. Attendants must be made to understand this, for until they do they are more apt to rule by brawn than by brains, thus adding to the patients' affliction.

There are many patients who quickly show the results of the slightest mistreatment by becoming irritable, violent or even so emotionally wrought up that they develop a convulsive seizure. In a cottage for small children there is a boy who finds pleasure in standing up in his chair playing with the window-blind string. The attendant does not want him to do this. So he observed that he could, by scolding and slapping the child's hands, compel him to sit quietly in the chair. This he would do, but with a scowl of unpleasantness on his face which would shortly be terminated by his falling in a seizure. He observed, also, that instead of this procedure, that by dangling a string from his pocket alongside of the window blind string that the boy would take this string with delight. Now, by fondling and caressing the little fellow in a gentle manner he could place him in the chair where he would play peaceably with the string for hours without having a seizure. This is but one example of the right and wrong way of managing the epileptic. When mistreated his condition is made all the worse, but when handled with judgment and understanding his attention is easily attracted from the undesirable to the desirable by proper suggestion. Although many of these children are crippled or feeble-minded, even to the extent that they are bedfast, yet they manifest a consciousness of feelings of a marked degree. They know when they are warm and when they are dry and clean. They know when

they are hungry, and are conscious of many things which concern their individual welfare which we fail to notice. They love to be petted and are very solicitous of any attention that may be given them. In this they are not unlike any child, unless we seek to find particular emotional differences. A normal child has its likes and dislikes. A newborn baby does not find things altogether pleasant in its new surroundings. It has been used, before it was born, to sharing the warmth of its mother's body, to obtaining its food and breath through her blood. When born it must face new conditions, in that it must breathe for itself and obtain its food in a new way. Likewise it must tolerate rough, irritating clothing in order to keep warm. If there is anyone who thinks this a pleasant experience he will have that idea divorced from his mind if he will but gaze on the wailing face of the baby just born. From beginning, then, until the end of life every person is confronted with the necessity of meeting unpleasant experiences which may be made less tolerable if proper judgment is not used.

Besides the unfavorable attitude of the patient toward interests outside of his own immediate desires we have many other conditions which aggravate unpleasant emotional states. Our records show more seizures on Sunday than on any other day of the week. Everyone recognizes this as the "longest day of the week," because of inactivity. Such a time affords an opportunity for worrying about getting home or just any thing which would have no place in a mind exercised with pleasant occupation. Likewise, more seizures occur during the "long winter days" than in the summer. Long-drawn faces, as seen in the epileptic, tell the story of the producing mental attitude. It becomes a problem for serious study, then, if we may make conditions good enough that we can help them to develop a habitual smile.

In conclusion, then, we must look upon epilepsy as an abnormal muscular reaction to strong mental states. It is an abnormal expression because such muscular activity does not gain the end for which the emotional state was generated. It is unnatural, also, since it is effort undirected. The epileptic, because of his peculiar make-up, cannot avoid the dangers of too great stress as the normal man meets it, but by an emotional drive that cannot be readily checked, labors on to mental exhaustion in unconsciousness. This is not deep enough to involve the motor or life centers of the brain, so a convulsion takes place. Viewing epilepsy in this light, we are now able, in treating the disorder, to institute more rational methods than has previously been had in surgical procedure and in empirical therapy.

PROGRESS
OF
MEDICAL SCIENCE

MEDICINE

UNDER THE CHARGE OF

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Note on the Presence of Meningococci in the Skin Petechiæ in Cerebrospinal Fever.—MUIR (*Jour. Royal Army Med. Corps*, London, 1919, xxxiii, 404) reports two instances in which death was preceded by cough and purulent sputum and by a petechial eruption. The smears from the petechiæ showed free and intraleukocytic Gram-negative diplococci. The same diplococcus was found in the blood but not in the cerebrospinal fluid, and there were no postmortem evidences of meningitis. The meningococcus belonged to Gordon's type II. These observations confirm the findings of others who, however, in some instances, thought the diplococcus differed from the usual meningococcus.

Epidemic Gastro-enteritis Due to Food Poisoning.—COMRIE and BIRD (*Jour. Royal Army Med. Corps*, London, 1919, xxxiii, 374) observed an epidemic of acute severe gastro-enteritis among British troops in July, 1918. There were in all about 400 affected troops. The symptoms were violent vomiting, frequent diarrhea, fever often to 102° F., subsiding to normal in three days. The stools were thin and bright green. Signs of circulatory failure were common in the severe cases. The epidemic was due to the use of warmed-over meat in "sea pies." In the fatal cases the Bacillus aertrycke (mutton strain) was found in the spleen. The same organism was found in the feces of six other cases. Treatment consisted in early catharsis followed by sedatives and cardiac stimulants. Saline infusions were tried in the very ill patients. A complete bacteriological and immunological study of the organism is presented.

SURGERY

UNDER THE CHARGE OF

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Treatment of Chronic Empyema.—TINKER and WATTENBERG (*Ann. Surg.*, 1919, lxx, 545) outline the results of their treatment of 80 cases of chronic empyema in the U. S. G. H. No. 26, at Fort Des Moines, Iowa. All but six of the cases had been operated on prior to their admission to the hospital. Seventy-two (90 per cent.) of their cases showed discharging sinuses from their pleural cavities. The remaining 8 cases had external wounds which had healed, but needed further treatment to develop their lung capacity and general physique. Of these 8 cases 3 reopened spontaneously soon after their admission. The majority of their cases of empyema followed influenzal pneumonia. Sixty cases had had only one rib resected, and they believe that the removal of more than one rib for drainage purposes is contra-indicated. The seventh or eighth rib was most commonly removed, the incision being made in the posterior axillary or midinfrascapular line. Of these 80 cases the length of time during which suppuration had existed prior to admission varied from twelve months to three weeks, with an average of four months. Forty of their cases were treated by the Carrel-Dakin method. The average time required for healing under this treatment was thirty-four days. Patients operated on from the eighth to the tenth day following the diagnosis of empyema progressed most rapidly. In no case did simple aspiration cure or even improve the condition. The Dakin irrigation was carried out routinely by a thorough flushing out of the whole cavity, once a day, with a liberal irrigation once every two hours. The tubes were so managed as to flush the entire cavity. In those cases which proved intractable to treatment bismuth paste was injected, hot, to fill the entire cavity, and the drainage tubes were permanently withdrawn. But 3 mild cases of bismuth-poisoning were observed in the 40 cases so treated. In addition to the local treatment, lung expansion was promoted by the use of Wolfe's bottles and by properly graduated outdoor setting-up exercises. It is believed that these results obtained from Dakin's treatment supplemented by bismuth paste when necessary, and aided by lung expansion exercises, setting up drills and a forced diet, show that a radical reoperation is unnecessary to effect a cure in the great majority of cases of chronic empyema.

Spontaneous Rupture of a Varicocele.—VAN DEN BRANDEN (*Arch. méd. Belges*, 1919, lxxii, 683) reports a case of spontaneous rupture of a varicocele which is a rare condition. Palet has compiled five cases, all occurring in old men. Van den Branden's case occurred in a young man

who experienced excruciating pain in the scrotum and fainted while shaving. Examination in the hospital disclosed an enlarged, tense scrotum. There was no history or evidence of venereal disease, but eight years previously the patient had been treated for an acute varicocele. Operation two days after admission revealed a hematocele, which was evacuated and drained. The wound healed by primary intention and the patient was discharged.

Personal Experience in the Use of Lane Plates.—SWEET (*Jour. Orthop. Surg.*, 1919, i, 673) reports 1560 fractures of long bones, with 172 open operations. Of these 41 were compound and 129 were cases of simple fractures, delayed or non-union, or were cases of vicious union. Lane plates were applied in 28 instances (femur 18, tibia and fibula 7, radius and ulna 3). Primary union occurred in all except 2 cases, and these were compound fractures. In 4 cases sinuses developed at the end of six to ten weeks, the process being afebrile. In 1 case in which the plates and screws were handled by the operator a low-grade osteomyelitis resulted. Sweet thinks operation is safer in the femur than in the other long bones, and considers operation highly satisfactory.

PEDIATRICS

UNDER THE CHARGE OF

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The Antiscorbutic Value of the Banana.—LEWIS (*Jour. Biol. Chemistry*, November, 1919) says that as a result of experiments he found that guinea-pigs fed an exclusive diet of banana were unable to maintain their bodily weight and died in from twenty to thirty days. He found in autopsy a condition of marked inanition, but no lesion characteristic of scurvy. Bananas in amounts greater than 25 grams a day as a supplement to a diet of rolled oats prevented the onset of scurvy. Such a diet did not permit normal growth in young animals. Less than 25 grams of bananas a day as a supplement to the oat diet did not protect against scurvy. Scurvy can be readily produced experimentally on a diet of autoclaved rolled oats supplemented by bran, milk casein and inorganic salts. When such a diet is supplemented by banana from 10 to 15 grams will serve to protect against scurvy, but result in rapid growth of young guinea-pigs. These experiments suggested that a lower content of the antiscorbutic principle may be sufficient to protect against scurvy, if the diet is adequate in its content of the other essential dietary constituents.

The Heat Coagulation of Milk.—SOMMER and HART (*Jour. Biol. Chemistry*, November, 1919) say that the chief factor in the heat coagulation of milk is the composition of the milk salts. Apparently casein

requires a definite optimum calcium content for its maximum stability. The calcium content of casein is largely controlled by the magnesium citrates and phosphates present. In fresh milk there is no relation between titratable acidity and heat coagulation. Acid fermentation in milk lowers the coagulability point by changing the reaction and by lowering the citric acid content. The titratable acidity of fresh milk samples varies so widely that it is impossible to determine the extent of acid fermentation by titration; so that it is impossible to use the acidity of milk as a criterion of coagulability. Difference in concentration accounts partly for the difference in coagulation of fresh milk samples. Hydrogen ion concentration is not the determining factor in fresh milk coagulation. It is nevertheless a factor in fresh milks and in commercial milk it may become an important factor.

The von Pirquet Test.—FRIEDMAN (*Colorado Medicine*, October, 1919) publishes the results of observations on 464 unselected patients. Their ages ranged from a few months to eighteen years. Of this number 219 were males and 245 were females. Of these 39.8 per cent. reacted positively, the number of positives among the males being 2 per cent. in excess to that found in the females. Under one year almost 12 per cent. reacted positively; from ten to fourteen years, 55 per cent.; and from fourteen to eighteen years, only 51 per cent. The highest number of positive reactions was noted during the eleventh to thirteenth year period, with 66.7 per cent.; and the lowest at the fifth to the sixth year period, with only 13 per cent. positive. Of 27 having a tuberculous parent 23.2 per cent. reacted. Of 181 children exposed to a parent with an active lesion 58 per cent. reacted positively, and of 78 cases exposed to a parent with a healed lesion, 38.5 per cent. reacted positively. Of those children whose father had a healed lesion, 48.4 per cent. reacted positively. Of 210 children examined from eighty-five families all members of the same family reacted positively in 55 instances. One hundred and eleven tests were performed upon 55 children at intervals of from several months to two and a half years. In 43 the outcome was the same with both tests. In 11 a negative result became in a subsequent test positive, and only once did a positive test become negative. In 6 children the first positive test was more pronounced, in 7 the second was more pronounced. In this series only 5 were found in whom clinical tuberculosis was demonstrable; 4 showed apical involvement and 1 has tuberculosis of the spine. None of these was under fourteen years of age; 4 were of tuberculous parentage; 3 reacted positively, but only 1 of these showed a strong reaction.

The Nervous Child and His Management.—MCCREADY (*Jour. Am. Med. Assn.*, October 11, 1919) points out some of the factors giving rise to the development of nervous disorders of later life. As regards treatment he suggests that this must begin with the modification of the immediate environment. The difficulty of improving in his own home is not because the home is essentially unfit but because it is difficult to eliminate the causes of the nervous disorder. Because of lack of special training the parents and relatives should be out of contact with the little patient. If the child is kept at home the domestic arrangements must be modified and changed as conditions require, sources of friction

must be removed, impatience, irritability and undue sympathy must be eliminated. Rest is the first essential. Next comes regulation of diet. Excessive carbohydrate consumption in the form of candy and pastry are to be eliminated. Fresh air, bathing, regulated exercise all have an important place in the hygienic management of the nervous child.

OBSTETRICS

UNDER THE CHARGE OF

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Transmission of Smallpox to the Fetus.—CAPPELANI (*Pedriatia*, Naples, April, 1919) observed four cases in which the newborn infants developed smallpox immediately after birth. At first these children seemed normal, but in five or six days smallpox developed. In one the mother was healthy but the infant had fever twenty-four hours after birth and developed smallpox the next day. The mother was revaccinated at once giving a typical response, but escaping smallpox, although the disease was prevalent at the time.

Cesarean Section for Hemorrhage from Varicose Veins of the Vagina.—BRUNNER (*Correspondenzblat f. schweiz Aerzte.*, March 15, 1919) has collected cases to show that hemorrhage from varices in the vagina during pregnancy is often fatal. In one collection of 15 cases there were 13 deaths, and during labor in a series of 16 cases there were 7 deaths. Another observer in 30 cases found 11 deaths and another in 3 cases had 2 deaths. In 1 case the obstetrician was able to arrest the bleeding by tamponing, but the moment the tampon was removed the blood spurted anew from various points. He delivered the patient by abdominal Cesarean section and the varices by the third day had collapsed. Brunner reports a case in which the child was very large, weighing 4900 grams. Profuse and persistent bleeding came on during labor and the labia were so edematous that ligatures would not hold and the hemorrhage was controlled by clamps. After abdominal section the woman recovered and her recovery was complicated by sepsis for which she received antistreptococcus serum.

Drugs Contra-indicated During Pregnancy.—MUNOS (*Repertarto de Medicina y Ciurgia*, Bogota, February, 1919) would not administer potassium iodid to pregnant women. He asserts that he has seen it induce abortion, killing the fetus in later pregnancy and even disturbed nursing infants when the drug had been applied externally to the breasts. Iodin acts comparatively like a stimulant with menstruation. Russian physicians have been in the habit of using iodin and potassium iodid extensively in typhus fever. In amenorrhea in robust

women iodide is useful and in the neuralgias of diabetics hypodermic injections of solution of potassium iodid often do good. These injections take the place of morphin. In albuminuria and nephritis morphin must be avoided.

GYNECOLOGY

UNDER THE CHARGE OF

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Large Doses of Radium in Cancer.—TURNER (*Lancet*, 1919, excvii, 1018) is of the belief that in treating cancer of the cervix with radium it is important to give at first a sufficiently large dose and he is accustomed to administer from 5 to 10 and even 15 thousand milligram hours and he states that he has never seen bad results follow such large doses. Suppose that the amount of radium employed in a particular case amounted to 100 mg., which is commonly the case, then a dose of 10,000 mg. hours would be administered by keeping it *in situ* for 100 hours. It might be suggested that it would be well to divide the exposure into two or more parts at intervals of a week or two, but Turner states that this will not answer and the whole dose should, if possible, be given at once. The patient should be kept in bed for a short period after exposure and should be douched. She may then return home but should report herself in about three months if all be going well, by which time it is possible that all the local evidences of the disease will have disappeared. Such dosage as Turner recommends is considerably in excess of the average dose of the American gynecologist for similar cases and it is very encouraging to note that such large doses can be given without causing extensive destruction of the surrounding tissues.

New Method of Uterine Suspension.—The permanent operative correction of a well marked uterine prolapse has always been one of the bugbears of gynecological surgery and therefore a new method of treatment which utilizes a strip of fascia lata, suggested by FREEMAN (*Surg., Gynec. and Obst.*, 1919, xxix, 511), may be of interest to those who have to treat such distressing conditions. Having obtained access to the abdominal cavity through a median suprapubic incision of sufficient size, the uterus is brought up into the opening and inspected. If the patient is still within the childbearing period, she must be sterilized, best by ligation of the tubes with silk, dividing them and perhaps folding the severed ends upon themselves. A strip of fascia lata, about six inches in length and three-fourths inch in width, is then obtained from the outer side of one of the thighs in the following manner: An incision of sufficient length is made through the skin and subcutaneous fat directly

down to but not through the glistening white fascia, and extending along the lateral aspect of the limb, midway between the trochanter and the knee. With a gauze covered finger the adipose tissue is pushed to either side so as to clear the field and the fascia divided in two parallel lines of the required length. The strip is then loosened and elevated by slipping the handle of a scalpel beneath it and sweeping the instrument from one end to the other. After dividing the attached ends with scissors the strip is removed and enveloped in moist gauze until required. The slit in the fascia lata from which the graft was taken may then be closed with a running suture of chromic gut, although this is not absolutely necessary because no harm will result if this is not done. Returning to the abdomen, the uterus is held firmly while a pair of small, sharp pointed, curved hemostatic forceps is plunged from one side to the other directly through the substance of the uterus close beneath the peritoneum covering the fundus, but not penetrating into the cavity. The forceps should be entered just internal to the attachment of one of the tubes and brought out at a corresponding point upon the opposite side, although if the organ is large it may be well to tunnel it somewhat more anteriorly in order to prevent undue pressure upon the bladder when the uterus is suspended from the tendons of the recti muscles. In order to facilitate the insertion of the forceps, it is occasionally desirable to nick the peritoneum with the point of a knife. When the forceps have been passed and are still in position, the fascia is doubled longitudinally upon itself, one end seized in the jaws of the instrument and the strip dragged through so that its center rests in the middle of the tunnel and its loose ends project from either side. Catgut stitches are then inserted so as to close the openings of the tunnel, thus preventing oozing and holding the fascia in place. The next step is to secure the ends of the fascia around the tendinous insertions of the recti muscles in order to bind the fundus of the uterus securely and closely to the anterior abdominal wall. This is accomplished first by stripping back the anterior sheaths of the muscles for a short distance above the pubes, so as to uncover the tendons, and plunging through these and the underlying peritoneum from without inward, a pair of pointed hemostatic forceps with which the ends of the fascial strip are seized and dragged into place, one on either side of the abdominal incision. After the peritoneum is closed, the ends of the fascial strip, which have been retained in forceps to prevent retraction, are pulled tight enough to hold the uterus firmly against the abdominal wall and are then crossed over the median line, best by tying them in a half-knot, and stitched securely to each other in several places by means of chromic gut so that they cannot slip. The wound is then closed in layers. Additional security against slipping may be obtained by catching the ends of the suspending fascia in the bight of a figure-of-eight silkworm-gut suture used in closing the abdominal incision. Fascia lata has a number of things in its favor: (1) It is easy to obtain in any desired quantity. (2) It is very strong and will not stretch to an appreciable extent, thus differing from the natural supporting ligaments of the uterus. (3) It does not become absorbed, like catgut, but incorporates itself within the tissues and permanently holds the uterus where it is placed.

PATHOLOGY AND BACTERIOLOGY

UNDER THE CHARGE OF
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A Comparison of Certain Antigens used in Complement-fixation Tests in Pulmonary Tuberculosis.—YOUNG and GIVLER (*Am. Rev. Tbc.*, 1919, iii, 476) compared the value of three antigens, the Petroff methyl alcohol soluble, the Corper autolysate and the Wilson bacillary. Their purpose was to find if the Wilson antigen was more suitable for complement-fixation tests than the others. This did not prove to be the case. Examining known cases of tuberculosis, they obtained best results with the Petroff antigen, obtaining 66 per cent. positives against 63 per cent. with Corper antigen and 57 per cent. with the Wilson antigen. They felt there was no justification for the use of the Wilson antigen, the bacilli emulsion having lost much of its antigenic properties by the alcoholic extraction in the preparation. They showed that when this extraction was carried further all of the antigenic properties are destroyed. They found the human hemolytic system superior to the sheep system in doing the tests. The serum of only 12 out of 75 guinea-pigs used proved unsuitable.

Toxic Necrosis and Regeneration of the Acinar Cells of the Pancreas.—Heretofore our knowledge of the action of various toxins upon the acinar cells of the pancreas with the occurrence of mitotic figures and evidences of regeneration has been quite meager. PARKER (*Jour. Med. Res.*, 1919, xl, 471) studied sections from some four hundred autopsies, of which one hundred showed lesions of necrosis in the acinar cells of the pancreas. All tissues were fixed in Zenker's fluid and stained by Mallory's eosin-methylene blue method. The author classifies the necroses of the pancreas etiologically into (1) acute pancreatitis with fat necrosis; (2) necrosis due to toxins; (3) infectious pancreatitis arising by direct extension along the ducts, from the blood stream or from a neighboring focus. In toxic necrosis the pancreas, macroscopically, shows no changes. Microscopically, necrosis may occur in a single acinar cell, groups of cells or diffusely, the total number varying considerably. As in other organs, the necrotic cells are quickly removed by polymorphonuclear and endothelial leukocytes, regeneration rapidly ensues and repair is complete. Inasmuch as similar lesions were found in the heart, liver, kidney and adrenals, because of the type of associated infection, by the fact that there was no involvement of the stroma and no demonstrable organisms, it is concluded that the necroses are of toxic origin. The necroses were most frequently encountered in pneumonia, diphtheria, acute peritonitis and other suppurative processes, due to streptococcus or pneumococcus. In a study of eighty-five cases it was found that the adrenals, pancreas, liver, kidney and heart showed toxic lesions most frequently in the

order named. Experimentally, a guinea-pig was subjected to chloroform vapor for two hours. Autopsy material showed active regeneration of liver, adrenal and pancreas, the latter exhibiting no remains of necrotic cells. Another guinea-pig was given liquor potasii arsenitis by mouth and died in five days. Sections showed fatty degeneration of the liver, but no necroses or mitoses, while the pancreas, kidney and particularly the adrenal showed no necroses but signs of active regeneration. The author concludes that these experiments confirm the findings in the human cases in that substances affecting the liver, kidney and adrenal affect the pancreas.

Studies in Epidemic Encephalitis.—LOEWE, HIRSHFELD and STRAUSS (*Jour. Infect. Dis.*, 1919, xxv, 378) report results of injection of monkeys, subdurally and rabbits intracranially, with filtrates of nasopharyngeal mucous membrane from cases of epidemic encephalitis. Lesions were produced in monkeys and rabbits similar to those found in the human brain in fatal cases of epidemic encephalitis. The virus was carried through four generations in rabbits, transmitted to a monkey in the fifth generation and then brought back to rabbits. A natural immunity was found in 50 per cent. of the rabbits. The virus was recovered from the nasopharynx of an animal inoculated intravenously. Cultures of the material used for inoculation were negative.

HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

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An Investigation of the Shaving Brush Industry, with Special Reference to Anthrax.—The Public Health Service (*Public Health Reports*, 1919, xxxiv, 994) makes a report on the shaving brush industry from the anthrax viewpoint, as a number of cases of facial anthrax have occurred from contaminated brushes. It appears that the anthrax spores are found almost exclusively on horse-hair imported from oriental sources, and various more or less efficient means of treating the hair were in use, but it was found that boiling or steaming of the hair offered the best means of destroying anthrax spores.

Fishes in Relation to Mosquito Control in Ponds.—HILDEBRAND (*Public Health Reports*, 1919, xxxiv, 1113), an ichthyologist of the U. S. Bureau of Fisheries, reports on the efficiency of top minnows, *Gambusia*

affinis, in the destruction of mosquito larvæ, with the object of aiding antimalarial work. The particular fish selected has the advantage of not requiring special environment for depositing eggs and hatching, as the young are borne well developed and will begin to destroy larvæ when only a day old. The top minnow must be protected from man, who uses them for bait, and from certain predatory fishes, notably black bass. Overgrowth of vegetation furnishes a natural protection for *Anopheles* larvæ which prevents the top minnows from reaching them. The author saw one top minnow eat 165 larvæ in less than twelve hours. The number of minnows required varies with local conditions; it is not possible to have too many. The author considers these fish valuable aids in antimosquito work and points out their limitations.

The Nature of Contaminations of Biological Products.—BENGTSON (*Hygienic Laboratory Bulletin No.* 112, U. S. Public Health Service, Washington, D. C.) found that the contaminations that occur accidentally in biological products were in general harmless for laboratory animals. Cocci and both spore-bearing and non-spore-bearing rods were present. An organism resembling the tetanus bacillus was found, but was without pathologic effects. Pathogenic contaminations are believed to be of animal origin and are chiefly found in products from animal sources while bacterial vaccines all proved to be contaminated with saprophytic forms.

Phenols as Preservatives of Antipneumococcic Serum. A Pharmacological Study.—VOEGTLIN (*Hygienic Laboratory Bulletin No.* 112, U. S. Public Health Service, Washington, D. C.) studied the effects of the administration intravenously to animals of mixtures of horse serum and phenol and trikresol, using doses that would contribute evidence as to the possibly deleterious action of the preservatives when the preserved serum is used in the treatment of pneumonia. The observations were made on normal animals and on those suffering from pneumonia. It was shown that certain toxic manifestations were less pronounced when a phenol-serum mixture was used than when the same amount of phenol was given in Ringer's or Locke's solution. The worker shows that phenol and trikresol may safely be used as preservatives of serums that are given in even such large doses as is the case with antipneumococcus serum and that the injections should be made at a slow rate.

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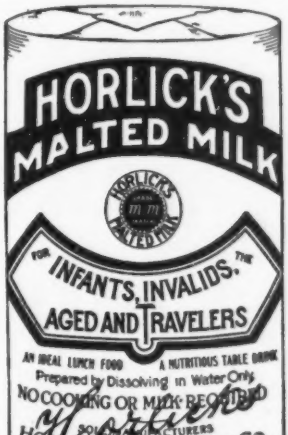
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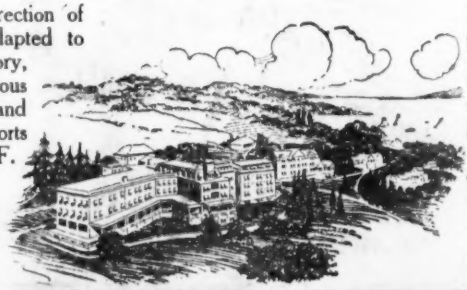

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